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Algemene Praktisynsreeks

DIAGNOSE VAN DIE ‚AKUTE BUIK'

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Die term ‚akute buik' is 'n vae benaming, maar behels tog enige buiktoestand wat as dringend beskou word, wat onmiddellik ondersoek moet word en wat dikwels 'n noodoperasie vereis.

Daar is geen vertakking van die chirurgie waar oordeel gebaseer op kliniese ondervinding van groter belang is, as die diagnose en hantering van akute siektes van die buik nie. Die bykomstige toetse wat in die afgelope jare ontwikkel is en wat so waardevol is by die diagnose van baie siektes, is grotendeels van min hulp in die akute buik. Die toepassing van moderne röntgenologiese metodes, alhoewel waardevol, is dikwels misleidend. Die allerbelangrikste faktore in die akute buikdiagnostiek bly nog steeds die neem van 'n noukeurige geskiedenis, sorgvuldige fisiese ondersoek, bloedtelling, en ondersoek van die urine.

Algemene praktisyns, veral die op die platteland, moet dikwels 'n belangrike besluit neem met betrekking tot die vervoer van die pasiënt na 'n sentrum waar die nodige hospitaalfasiliteite bestaan. En om die saak verder te bemoeilik bestaan daar, afgesien van verskeie chirurgiese toestande, ook 'n groot aantal interne toestande wat die diagnose kompliseer.

Die volgende is 'n lys van algemene chirurgiese buiktoestande wat 'n noodoperasie mag vereis:

1. *Beserings*
   (*a*) Penetrerende en perforerende wonde.
   (*b*) Indirekte beserings en kneusings.
2. *Infeksies*
   (*a*) Appendisitis en divertikulitis.
   (*b*) Peritonitis na perforasie. In hierdie verband moet spesiale vermelding gemaak word van (i) peptiese ulsers, hetsy van die maag of duodenum; (ii) vreemde voorwerpe soos visgrate, hoenderbene, spelde, ens.; en (iii) Richter se hernia.
   (*c*) Cholesistitis met komplikasies—empieem, gangreen, peri-cholesistitis, peritonitis.
3. *Dermobstruksie*
   (*a*) *Die kleinderm:* (i) eenvoudige obstruksie, en (ii) beklemming.
   (*b*) Die grootderm: (i) eenvoudige obstruksie, (ii) beklemming, en (iii) volvulus.
4. *Mesenteriese Trombose en Embolisme*
5. *Bloeding*
   (*a*) Gastro-intestinaal.
   (*b*) Intraperitoneaal, byvoorbeeld in miltskeur.
   (*c*) Retroperitoneaal, byvoorbeeld 'n nierskeur.
   (*d*) Postoperatief.

AKKURATE GESKIEDENIS

Die benadering van 'n akute buikgeval behels, eerstens die neem van 'n akkurate geskiedenis. Die volgende vrae moet in hierdie verband gestel word:

1. *Wanneer het die pyn begin?* Was dit gedurende die dag of nag? Hoe laat? Hoe was die kronologiese verloop van die siektebeeld—stap vir stap. Het die pyn ingetree na 'n groot maaltyd of na die gebruik van 'n oormaat alkohol? Perforasie van 'n ulkus van die maag of duodenum en perforasie van die esofagus, asook 'n akute pancreatitis tree dikwels na 'n maaltyd in.

2. *Waar het die pyn eerste ontstaan in die buik?* Dit is van hulp as die pasiënt self aandui waar die pyn begin het, indien moontlik met een vinger.

3. *Waarheen het die pyn gegaan of gesprei?* Die klassieke geskiedenis van die pyn van akute appendisitis met sy oorsprong in die middel van die buik of bobuik, met verspreiding na die regter onderbuik, word beskrywe. Die pyn kan egter ook in die regter onderbuik, of in die onderbuik ontstaan. Die klassieke beeld van pyn wat in die galblaas of galbuis ontstaan, is aan ons bekend, maar dié soort pyn kan ook atipies voorkom. Byvoorbeeld, dit kan in die epigastrium ontstaan en dan links oor die bobuik na die rug of linkerblad en skouer, en af in die arms versprei, m.a.w. dit kan die pyn van 'n koronêre trombose of spasme naboots.

Dunderm-pyn, byvoorbeeld die pyn wat ontstaan as gevolg van 'n dundermobstruksie is gewoonlik in die middel van die buik gelokaliseer, en dit het die tipiese geaardheid van koliek. Pyn in die bobuik wat deur gaan na die middel van die rug toe, ontstaan gewoonlik vanaf retroperitoneale organe, soos byvoorbeeld, die pankreas, die niere, en die aorta. Dink maar aan die rugpyn waaroor die pasiënt kla wat 'n penetrasie van die maag of duodenale ulkus gehad het. Pyn, hoog in die epigastrium naby die xifisternum, wat pre-sternaal versprei, word dikwels deur 'n maagulkus hoog op in die klein boog van die maag of ondereinde van die esofagus veroorsaak, soos byvoorbeeld in die geval van esofagitis wat veroorsaak word deur terugvloeiing.

4. *Wat is die geaardheid van die pyn?* Is dit 'n koliekpyn, m.a.w. is die pyn krampaardig met tussenposes van verligting? 'n Koliekpyn ontstaan as gevolg van die een of ander obstruksie in 'n hol orgaan, soos byvoorbeeld in die maagdermkanaal, ureter, galbuisie of galblaas. Dit is kenmerkend van hierdie soort pyn dat die pasiënt rusteloos is en dat hy groot moeite ondervind om doodstil te lê; ook dat die pyn van 'n kwaai gehalte is en groot dosisse verdowingsmiddels vereis om dit te verlig.

DIE BYVERSKYNSELS

Het braking, diaree, hardlywigheid of geelsug ingetree? Refleksiewe braking word gevind by akute cholesistitis, galsteenkoliek, akute appendisitis, nierkoliek akute pankreatitis, akute piëlosistitis, asook by sekere ginekologiese toestande soos byvoorbeeld steeldraai van 'n sis van die ovarium. Hierdie braking is gewoonlik 'n kort episode. As dit aanhoudend is moet meer spesifiek aan gastro-intestinale toestande gedink word, soos byvoorbeeld, gastro-enteritis en obstruksietoestande van die maagdremkanaal. Hoe hoër die obstruksie voorkom, hoe erger en meer aanhoudend is hierdie simptoom. Daar moet op die aard van die braaksel gelet word; vars bloed, die koffiemoer-voorkoms van ou bloed, bestanddele van 'n onlangse maaltyd, of maaginhoud van 'n dag of wat gelede as gevolg van obstruksie van die pilorus. Verder is die stinkende troebel braaksel van dundermobstruksie kenmerkend. By klein babetjies is dit allerbelangriks om vas te stel of daar gal in die braaksel is of nie, want in dié bevinding lê die geheim van die differensiële diagnose tussen 'n hipertrofiese pilorus-stenose en aangebore duodenale obstruksie, opgesluit.

Pertinente vrae aangaande die pasiënt se vroeëre gesondheidstoestand en gewoontes moet altyd aan die pasiënt met 'n akute toestand van die buik gestel word. Besonderhede aangaande dispepsie, slukmoeilikheid, eetlus, gewigsverlies, ontlastingsgewoontes, swart stoelgange, hardlywigheid, geelsug, teenwoordigheid van bloed en slym in die stoelgang, die aan- of afwesigheid van 'n breuk en vorige operasies is van belang. Aangesien die bejaarde pasiënt met 'n akute buik dikwels 'n vergrote prostaat het, moet navraag gedoen word na verskynsels van prostatisme.

ONDERSOEK VAN DIE PASIËNT

Daar moet op die algemene voorkoms van die pasiënt gelet word. Is die pasiënt rusteloos of lê hy doodstil in die bed? Wat is die voorkoms van die vel en slymvliese met betrekking tot die aan- of afwesigheid van anemie? Is dehidrasie teenwoordig? Is die pasiënt geelsugtig? Dit is moeilik om geringe mate van geelsug by elektriese lig vas te stel.

Die neem van die pols met betrekking tot snelheid en gehalte is vanselfsprekend, maar daar is tog geneeshere wat dit nie doen nie. Die bloeddrukbepaling is absoluut noodsaaklik in elke geval. 'n Ernstige skoktoestand met verlaging van die bloeddruk bring ons op die spoor van 'n akute mesinteriese trombose, beklemming van 'n dermlus, akute hemorragiese pankreasnekrose, 'n akute diffuse peritonitis of inwendige bloeding. Die herhaalde bepaling van die bloeddruk is 'n goeie aanduiding van hoe 'n geskokte pasiënt reageer op behandeling.

In babas en maer persone is peristalse dikwels waarneembaar in dermobstruksie. Dink maar aan hipertrofiese pilorusstenose en die verskillende aangebore obstruksies in die duodenum.

Betasting van die buik is onontbeerlik. Die plankharde onbeweeglike buikwand van 'n akute diffuse peritonitis, soos byvoorbeeld wat ons vind by 'n onlangse perforasie van 'n peptiese ulkus, is aan almal bekend. Sodra so 'n perforasie egter net bietjies-bietjies lek is die diagnose hiervan gladnie so duidelik nie en kan dit verwar word met akute cholesistitis, pankreatitis of akute appendisitis. Baie buike is al vir akute appendisitis oopgemaak waar dan 'n lekkende peptiese ulkus gevind is. Betas die buik om vas te stel waar die maksimale lokale drukteerheid geleë is. Toestande soos akute cholesistitis, akute appendisitis, akute pankreatitis, beklemming van 'n dermlus, of divertikulitis van die kolon moet in hierdie verband in gedagte gehou word.

As daar nog gepaardgaande lokale stywigheid waarneembaar is, dan is die parietale peritoneum by die lokale siektetoestand betrokke en dui dit op verspreiding van die infeksie of die perforasie. Die skielike loslaat van die buikwand met skielike subjektiewe pyntoename is hier ook van belang.

By babas is die betasting van die harde ovaalvormige tumor van 'n hipertrofiese pilorus-stenose in die regter bobuik absoluut diagnosties, so ook die betasting van die worsvormige tumor van 'n intussussepsie by jong kinders.

Hiperperistalse kan met die stetoskoop op die buik vasgestel word; gepaard met afwisselende pynaanvalle dui dit op moontlike dermobstruksie, maar daar moet onthou word dat erge gastro-enteritis presies dieselfde bevinding kan gee. Muntstukperkussie van die buik is van waarde om uitgesette dermlusse vas te stel. Dit word nou algemeen aanvaar dat die sogenaamde ‚kliesma-toets' by dermobstruksie waardeloos is, al word die kliesma herhaal, aangesien die pasiënt tog die kolon kan ontledig in so 'n obstruksietoestand. Daar moet nie vergeet word om die liesstreke vir 'n moontlike beklemde breuk te ondersoek nie en veral by die vroulike pasiënte moet 'n femorale breuk uitgeskakel word.

Rektale ondersoek is 'n vereiste by die pasiënt met 'n akute toestand van die buik en gee waardevolle inligting, soos bv. die teenwoordigheid van 'n karsinoom of poliep in die rektum, en bloed en slym in die rektum. Verder moet die toestand van die prostaat by die man en die toestand van die uterus en adnexa by die vrou ondersoek word. Tumore kan in die sak van Douglas gevoel word asook die teenwoordigheid van drukteerheid of 'n massa wat dui op infektiewe toestande, soos 'n bekkengeleë appendisitis of sigmoied divertikulitis. In die jong kind kan die voorstotende punt van 'n intussussepsie in gevorderde gevalle vasgestel word en die bekende pruimkonfyt-voorkoms van die stoelgang op die vinger gesien word.

As die diagnose van die akute buik onseker is, dan is dit 'n goeie beleid om na 'n uur of twee die ondersoek te herhaal en weereens te herhaal. Dit is tog verbasend hoedat akute buiktoestande, veral infektiewe letsels, hulself met die loop van tyd lokaliseer en die diagnose duideliker ontwikkel.

Daar moet onthou word dat die toediening van verdowingsmiddels soos omnopon, morfien of petidien by hierdie pasiënte lei tot verdwyning van die simptome. Indien die diagnose nog nie duidelik is nie, of as daar nog nie besluit is om 'n laparotomie te doen nie, moet verdowingsmiddels toegedien word met inagneming van hierdie feit.

Bykomstige Ondersoeke en Toetse

Die volgende bykomstige ondersoeke en toetse is ook van belang:

(*a*) *Ondersoek van die urine.*

(*b*) *Bloedtelling.* Die belangrike punte hier is die aanwesigheid van anemie, leukositose of leukopenie, 'n verskuiwing van die witselle na links of na regs, en die uitskakeling van een of ander retikulose.

(*c*) *Bepaling van die bloedureum.* Uremie kan die akute buik naboots en by die bejaarde man kan 'n bykomende hipertrofie van die prostaat die hantering van 'n buiktoestand bemoeilik.

(*d*) *Bepaling van die serum amilase.* Hierdie bepaling word gedoen in verdagte gevalle van akute pankreatitis, maar af en toe kan die waardes hier binne normale perke wees. Daar moet egter onthou word dat die serum amilase in perforasietoestande van die bobuik en in dundermobstruksie verhoog word. 'n Duidelike styging van die serum amilase na 300-400 eenhede sal egter die kliniese diagnose van 'n akute pankreatitis bevestig.

(*e*) *Röntgenondersoek van die buik.* Die diagnose van die akute buik moet nie staan of val by die röntgenondersoeke nie en in elke geval moet die hele siektebeeld in aanmerking geneem word voordat die finale diagnose gestel word.

GYNAECOLOGICAL AND OBSTETRICAL CAUSES OF ACUTE ABDOMINAL OR PELVIC PAIN

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The object of my contribution will be to enumerate only the commoner gynaecological and obstetrical conditions which may confuse the general practitioner in the diagnosis of a case presenting with abdominal or pelvic pain. No attempt will be made to discuss each condition fully. Mention will only be made of certain aspects which may be helpful in arriving at the correct diagnosis.

1. ECTOPIC PREGNANCY

These patients present clinically either as acute, or as subacute and chronic cases.

Acute Cases

It must be remembered that only about one-third of ectopic pregnancies will present themselves as acute surgical emergencies. In this group we include 3 clinical types:

(*a*) *Ruptured Ectopic Pregnancy*

This is the classical case we were impressed with at medical school, which is associated with a sudden onset of acute lower abdominal pain, collapse and shock. The clinical picture is that of severe internal haemorrhage. The pain rapidly involves the entire abdomen and palpation reveals the classical signs of distension, marked rebound tenderness, and shifting dullness in the flanks.

(*b*) *Leaking Tube*

Here the picture is less dramatic in onset, but still acute because of the active intraperitoneal bleeding, which may have been going on for periods of hours or even a day or two. The patient experiences several attacks of acute pain of a cramp-like nature, with a persistent dull ache between the acute episodes. The onset may simulate the more acute case but soon settle into the above picture. There may be vomiting and also scapular pain to confuse the issue. The scapular pain is due to irritation of the diaphragm by the blood in the peritoneal cavity. The patient also complains of fainting and weakness.

Examination of the abdomen reveals distension, rebound tenderness and shifting dullness. The tenderness is marked. On vaginal examination one may find an indefinite swelling in one or other fornix, but the pathognomonic sign is the severe pain caused by flipping the cervix with the examining finger.

Points to remember, in differentiating an acute ectopic from an acute pelvic infection, are as follows:

(i) In an ectopic the temperature is usually not raised.

(ii) There usually is no rigidity of the abdominal muscles in an ectopic. This is a very important sign.

(iii) The tenderness caused by movement of the cervix is much more acute in an ectopic. These patients almost climb out of the bed when their cervices are touched.

(*c*) *Unruptured Pregnant Tube*

This is mentioned for the sake of completeness, but as the condition is usually discovered accidentally, it will not be discussed. Although not an acute abdomen symptomatically, it should be operated on as an emergency.

Subacute and Chronic Cases

The subacute and chronic cases comprise more than one-half of all cases of ectopic pregnancies encountered. These patients do not present with acute abdomen, but will come to your rooms complaining of a dull ache or dragging pain in one or other iliac fossa or a continuous cramp-like lower abdominal pain. Often they have rectal tenesmus or difficulty with micturition, including retention of urine. The menstrual history usually includes a period of amenorrhoea, followed by continuous vaginal bleeding up to 3 weeks or longer. The history of the passing of a decidual cast is often mistaken for the aborting of products of conception.

There is an elevation of pulse and temperature, and slight pallor of the mucous membranes. Abdominal examination may show an indefinite mass arising out of the pelvis. This is due to matted omentum and bowel covering the pelvic haematocele. Bimanual examination reveals a tender, doughy swelling in one or other fornix, or in the pouch of Douglas. This mass is characteristically irregular in consistency, being hard in some and softer in other areas.

Particularly in the Bantu, it is often extremely difficult to differentiate such cases of tubal mole, with or without the formation of pelvic haematocele, from chronic pelvic inflammatory processes. When in doubt, take the case history again, paying particular attention to the menstrual history. A lowered haemoglobin concentration of the blood will point to a pelvic haematocele. Lastly, never forget the value of an examination under anaesthesia and the ease with which a colpotomy puncture can be performed. The blood aspirated from the peritoneal cavity or haematocele in such a case, is typical in appearance. Firstly, it is haemolysed blood and hence will not clot if left in a tube. Secondly, it contains numerous minute clots, as can be readily seen if squirted onto a clean piece of gauze. Blood aspirated from a vessel will not exhibit these characteristics.

OTHER CONDITIONS

2. *Acute Salpingitis and Rupture or Leakage of a Pyosalpinx*

Here again the taking of a proper case history will help immensely in arriving at the correct diagnosis. An acute attack of salpingitis nearly always follows within a few days after the menstrual period. The invading organisms are usually held up at the cervical barrier until menstruation

occurs, when they ascend by continuity of tissue to the fallopian tubes.

A vaginal discharge and bladder symptoms of dysuria—usually frequency and urgency of micturition—must accompany the condition. The patient has rigors and may vomit. The temperature varies from 101 to 103°F. The abdomen is distended, but now rigidity of the abdominal muscles is characteristic. An abdominal swelling arising out of the pelvis may be due to a hydro- or pyosalpinx. Vaginal examination may reveal a retort-shaped adnexal mass. Movement of the cervix, although tender, is not so excruciating as in the ectopic.

When a rupture or leakage of a pyosalpinx happens, there is a sudden deterioration in the condition of the patient, with the signs and symptoms of generalized peritonitis. There will be a sudden exacerbation in the abdominal pain and, on examination, the abdomen will be rigid and distended and at a later stage ileus may be present.

3. *Complications of Ovarian Cyst or Tumour*

The only time that an ovarian cyst or tumour causes acute abdominal symptoms is when it becomes complicated by torsion or rupture or a sudden haemorrhage takes place into it. It is well to remember that during pregnancy even the smaller cysts may undergo axial rotation, and the size of the cyst undergoing torsion has no bearing on the severity of the symptoms experienced. There is a sudden onset of acute lower abdominal pain of a cramp-like nature, and the other symptoms and signs of peritoneal irritation quickly follow. Characteristically, there is vomiting. The diagnosis is facilitated by not neglecting to do a vaginal or at least a rectal examination. A tumour or cyst can usually be found. A sudden increase in the known size of a tumour or cyst will indicate a haemorrhage into it, often as a result of torsion.

4. *Threatened or Septic Abortion*

These conditions should be easy to diagnose, but often a threatened, and especially a septic, abortion may cause acute lower abdominal cramp-like pains of such severity that it may quite easily be confused with an ectopic pregnancy or other lower abdominal emergencies. The patient who has a septic abortion is unlikely to mention to the doctor that she may have been pregnant. In fact, she will probably give a fictitious menstrual history and strongly deny the procurement of an abortion.

5. *Necrobiosis or Red Degeneration in Fibroids*

This complication nearly always occurs during pregnancy, postpartal or postabortal. It causes pain, which may occasionally be quite severe, and marked tenderness over the lower abdomen, and it is accompanied by pyrexia and leucocytosis. Particularly in the more advanced pregnant state, it may be difficult to distinguish from an extra-uterine cause. The condition usually subsides within a few days to a week, but it often is recurrent. No active treatment is required and to operate is extremely unwise and foolish.

Under this heading we can also include the pedunculated fibroid, which in rare cases undergoes torsion and causes acute symptoms.

6. *Ovulatory Pain (Mittelschmertz)*

In spite of the fact that this condition has been known to medical science for a hundred years, many an appendix has been removed and will probably still be removed in future, because practitioners do not take an adequate history. In the female this should always include the menstrual history.

Ovulatory pain occurs from time to time in approximately 15 - 40% of women. The pain is acute and may be severe, but seldom lasts longer than 12 - 24 hours. It is usually situated in the hypogastrium or one or other iliac fossa. Very often the pain occurs in the right iliac fossa, when the patient will be fortunate indeed if she manages to bypass the operating theatre. The time of onset is always on, or about, the 14th day before the onset of the next menstrual period. Only when a woman menstruates regularly every 28 days, will it occur in mid-cycle.

Two causes of the pain have been suggested. One is irritation of the blood and liquor folliculi on the peritoneum, when the pain will follow ovulation. The other theory is that the pain is caused by the increasing tension in the ovary as the follicle ripens. The pain would then occur before ovulation takes place. This may be why ovulatory pain seems to be commoner in sclerocystic ovaries, or where there is pelvic congestion.

CONCLUSION

This concludes the brief survey of the commoner gynaecological and obstetrical conditions which may confuse the issue in the diagnosis of the acute abdomen. Physical findings are seldom missed, but are often misinterpreted because not enough time is devoted to the interrogation of the patient and the sorting out of the episodes of the illness into their proper chronological order.

FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

Sixth International Congress on Diseases of the Chest. This Congress will be held in Vienna on 28 August - 1 September 1960 and information concerning the arrangements were published in this *Journal* last year (**33**, 599, 18 July). The Organizing Committee of the Congress have now forwarded a limited supply of brochures to the *Journal* office. These brochures contain full details of the programme, etc. and also application cards for participation and accommodation. Those wishing to obtain brochures should write to P.O. Box 643, Cape Town.

The Eighth International Congress of Blood Transfusion will take place in Tokyo, Japan, on 12 - 15 September 1960, as the 8th Congress of the International Society of Blood Transfusion. This is the first international scientific meeting on blood transfusion held in Asia. It will be preceded by the *8th International Congress of Haematology* to be held in Tokyo on 4 - 10 September 1960 in which 'Immunohaematology' will be one of the major subjects. The two Congresses will organize a joint symposium on 'Bone-marrow transfusion' on 12 September. The other main subjects of the Blood Transfusion Congress will be 'Extra-corporeal circulation', 'New aspects of blood groups (*a*) genetics, (*b*) serology, (*c*) blood groups and diseases, and (*d*) transfusions (including transfusion reactions to various antibodies)' and 'Biochemical and clinical aspects of blood proteins and new plasma fractions'. Preservation of blood cells and problems of the organization of a blood donor service, etc. will also be discussed during the Congress. Further information may be obtained from the Secretary-General, Dr. Seizo Murakami, Blood Transfusion Research Laboratory, Japanese Red Cross Society, Shibuya, Tokyo.

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EDITORIAL : VAN DIE REDAKSIE

FIBRINOLYSIS

If normal blood is diluted in a suitable buffered medium and then clotted, the clots can be observed to disappear over a period of hours. This phenomenon may be seen either in blood or plasma. The clot, which has a solid appearance, starts to fragment and ultimately lyses completely. This is due to the fact that normal human and animal plasma and serum contain a globulin, plasminogen which, in the presence of activators, is rapidly converted to plasmin, a proteolytic enzyme. This enzyme is able to attack various substrates such as gelatin, casein, fibrinogen and most important of all—fibrin. It is present in the blood in such a concentration that when fully activated it would be capable of digesting the total fibrinogen of the body in a few minutes.

Acute and chronic thrombo-embolic vascular disease is an important cause of human illness, and the possibility of its treatment by measures designed to produce dissolution of the causative thrombus or embolus by enzymatic means arouses exciting possibilities. Cliffton[1] has now gone so far as to say that the rapid dissolution of intravascular clots or thrombi by fibrinolytic activity is an accomplished fact. Progress in this field has been rapid as was clearly demonstrated[1] in a recent number of the journal *Angiology*.

In vitro experiments have shown that fibrinolytic activity can be induced in the blood by a number of different processes. Normal plasma, for instance, when treated with bacterial filtrates, develops powerful fibrinolytic and proteolytic activity. Streptokinase (SK), which is the name given to an extracellular product of haemolytic streptococcal metabolism, is capable of activating precursor substances in plasma and inducing fibrinolysis. It has thus been used in the lysis of extravascular fibrin clots in man, e.g. in fibrinous pleural effusions. However, the intravascular use of SK and SK-activated substances raises some difficulties.

The physiological process by which the enzyme becomes available for action *in vivo* is still in dispute. There are two main theories. Both agree that a pro-enzyme, plasminogen, exists in the plasma and this is activated to plasmin which is the proteolytic (fibrinolytic) enzyme. According to one theory SK or a similarly acting substance activates plasminogen to plasmin while additional SK reacts with plasmin to form an activator complex. Alternatively it is postulated that there is already a pro-activator present which combines with SK to form activator, and this activator combines with plasminogen to form plasmin. The dispute centres round the question: does SK activate or is a SK=enzyme combination the activator?

Naturally such a potentially powerful system could not be expected to exist in the blood without an equally powerful system of inhibitors; this proteolytic system needs to be uninhibited or 'free' if it is to act. It may be interfered with at least at three levels:[2] (1) SK may be neutralized by its specific antibody and so be unavailable for activity, (2) the activator may be neutralized by an inhibitor in the plasma, and (3) plasmin itself may be inhibited by another plasma substance.

The therapeutic applications of all this work is still in its infancy. There are at least two ways in which fibrinolysis may be stimulated. Either plasminogen of plasma can be activated to plasmin *in vitro*, purified, concentrated, and then injected, or else the patient's own fibrinolytic system can be activated *in vivo*. A number of studies relating to both types of approach to this problem have been recorded in the literature. For example, commercially available plasmin from streptokinase-streptodornase (SK-SD) activated human plasminogen has been injected intravenously in patients with venous thrombosis, arterial thrombosis and thrombosis of the central retinal vein.[1,3-7] The results are better in the treatment of venous thrombosis than in the treatment of arterial thrombosis, but improvement has occurred in both. Unfortunately, side-reactions, e.g. fever, chills, cyanosis, hypotension, leucopenia and thrombocytopenia occur. Some of these have been controlled by antipyretic and antihistaminic drugs and may be due to excess of SK-SD. The side-effects can be diminished by using less activator or by the preparation of more highly purified extracts, and have not been a real bar to extensive use of this preparation.

The patient's own fibrinolytic system has been activated by the injection of protein-free pyrogenic lipopolysaccharides.[8] Results were 'encouraging' especially in cases of venous thrombosis, but once again there were unpleasant and severe side-effects not completely controlled by antipyretics. Alternatively, a purified form of SK has been injected intravenously.[2,9,10] It was found that the dose of SK used was critical if re-formation of the clot was to be avoided. As a preliminary step it was necessary to give an initial, or priming dose just sufficient to neutralize the circulating SK antibody and inhibitor. Unfortunately the amount of these inhibitors varied considerably from patient to patient. Once neutralization had been achieved the use of different quantities of SK had different effects. Small amounts of SK resulted in moderate or large amounts of circulating plasmin. This resulted in a marked lengthening of the prothrombin time and depletion of fibrinogen with potentially serious effects to the patient. After the use of large amounts of SK, free SK remained in the plasma and there was depletion of plasminogen and in some cases actual re-formation of a previously lysed clot. The best results were obtained when the SK dosage was so adjusted as to generate small amounts of both SK and plasmin in the circulating blood. In this way consistent and reproducible intravascular clot lysis could be produced and re-formation of the clot did not occur. Side-reactions did occur but these were not unduly troublesome.

All this work points to a potentially powerful therapeutic tool. It is clear that the techniques are not as yet sufficiently standardized for general application. The use of fibrinolytic enzymes intravascularly, except under carefully controlled conditions, might be dangerous and should not yet be recommended. Results so far have been far better in the treatment of venous rather than arterial thromboses and have been better in the first 48 hours after the formation

of thrombus than subsequently. The treatment may be much more hazardous than the disease and there is evidence that immune responses may develop which might preclude further treatment along these lines. These principles cannot yet be successfully applied on a large scale in the treatment of such conditions as coronary or cerebral atheroma, but this type of treatment may not be long delayed.

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'N HOSPITAAL SONDER GERASE

Dit is interessant om daarop te let hoedat, by die opstel van die beginsels van hospitaal-beplanning en -konstruksie, die probleem van gerase in die verlede verwaarloos is. Van al die klagtes wat pasiënte oor hospitale opper (en dit is ook waar van goedgesinde, welwillende pasiënte wat in spesiale hospitale en opleidingshospitale verpleeg word) is dié oor gerase seker die mees konstante. Die soort gerase wat die meeste voorkom en die grootste ongerief veroorsaak vir pasiënte wat ernstig siek is, is die volgende:

Daar is, om mee te begin, gerase wat ontstaan as gevolg van die interkommunikasiestelsel in die hospitaal. By sommige hospitale is die skrikwekkende uitsaai-stelsel nog in gebruik. Dwarsdeur die dag en nag word die name van dokters wat êrens benodig word op so 'n manier uitgeroep dat daar geen twyfel kan bestaan oor die feit dat die geraas—want vir die pasiënt is dit niks anders nie—tot in elke hoekie deurdring. Oor die meriete van die verskillende soorte kommunikasiestelsels wil ons ons nie nou uitlaat nie. Dit is alreeds by 'n vorige geleentheid bespreek.[1] Wat ons egter wil benadruk is dat hierdie soort nimmereindigende geraas sonder twyfel 'n negatiewe en versteurende faktor is.

As deel van die kommunikasiestelsel is daar ook die telefoon. Pasiënte wat dit so ongelukkig tref om 'n kamer te hê iewers in die nabyheid van 'n telefoon, word dikwels dwarsdeur die dag en nag aan die luigeluid blootgestel, sowel as aan die gepraat van persone wat die telefoon gebruik.

Meganiese hulpmiddels in die hospitaal, soos byvoorbeeld hysers, stootstoele, trollies, apparaat, en kombuisgereedskap is verantwoordelik vir 'n groot deel van die versteurende geraas wat vir pasiënte met hoofpyn of prikkelbaarheid soms ondraaglik word. In sommige hospitale met 'n ouere soort uitrusting op hierdie gebied, vorm die geklap en gekrys van hyserbakke en deure so 'n ontstellende geraas dat dit nouliks denkbaar is dat dit deur die hospitaaloutoriteite gedoog word.

Onverantwoordelike geskerts en gepraat en gelag deur lede van die verplegingspersoneel sowel as deur lede van die publiek dra dikwels by tot die las van gerase waarmee die pasiënt opgesaal word. Ook is hospitale dikwels (veral in die grotere stede) op plekke gebou waar die verkeersgerase onvermydelik 'n versteurende element vorm.

Die meeste van die gerase waarna ons hierbo verwys het, kan deur goeie beplanning uitgeskakel word of deur goeie administrasie voorkom word. Aan die Universiteit van Münster in Wesfalië is byvoorbeeld onlangs 'n modelhospitaal gebou wat geheel en al klankdig is. Die hospitaal is so gebou dat alle soorte klanke geabsorbeer word. Alle moontlike bronne van gerase is by die beplanning en konstruksie van die hospitaal in ag geneem sodat die uiteindelike produk as 'n tegniese en argitektoniese model beskou kan word.

Dit is weliswaar die geval dat hierdie hospitaal, wat eintlik die oor-, neus- en keelafdeling van die Universiteit van Münster is, ontwerp is met die oog op spesiale oornavorsing. Ook het dit 'n groot som geld gekos om te bou en dit het drie en 'n half jaar geneem om die gebou te voltooi.

Daar kan nie orals oor die wêreld modelhospitale van hierdie aard gebou word nie. Maar, en dit is hierdie aspek van die probleem van hospitaalbehandeling wat ons hier wil beklemtoon, 'n voorbeeld soos hierdie dui op wat wel gedoen kan word om die gerief en die geluk van die pasiënt te verseker, bowe en behalwe die spesifieke mediese behandeling wat hy kry. Dat elke pasiënt nie net 'n geval is nie, maar ook 'n mens, met al die gewone menslike swakhede en met 'n vermeerderde prikkelbaarheid gedurende tye van ernstige siekte, kan nooit sterk genoeg beklemtoon word nie.

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THE SURGICAL TREATMENT OF AORTIC STENOSIS*

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In this short presentation I shall discuss the following features of aortic stenosis: (1) The basic indications for operation, (2) certain features of diagnosis, and (3) the type of operation to be performed and the results.

1. INDICATIONS FOR OPERATION

I have written and spoken extensively upon this matter of the need for operation in aortic stenosis[1] but, at the risk of being tedious, I shall make some comments.

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The attitude to aortic stenosis seems still predominantly to be governed by a mixture of complacency and of fear. We still hear the problem presented as that of a patient, usually a man, in the fifties or sixties who, having had a heart murmer for many years with trivial or no symptoms, develops symptoms of varying urgency. From this it is still argued that it is proper, at any age, to procrastinate because the patient may similarly live for many decades.

This is quite false, for aortic stenosis is a crippling and killing disease even in the very young. In children and in

adolescents it is particularly dangerous because of the simple fact that their young, strong heart muscle can over-exert itself abundantly and not show any symptoms of the strain to which it is constantly submitted. In this very tolerance lies the greatest danger, because when the muscle fails it fails rapidly. Sudden death is quite common; it is usually preceded by a period of the urgent features of left ventricular failure, but not always.

Similarly, it is my experience that in the twenties and thirties when clinical symptoms appear the disease is far advanced and if life is to be saved operation must be advised and performed promptly.

The complacency of the past is today often fostered by fear of the dangers of operation. This attitude is quite indefensible, for the medical mortality is very high, sooner or later approaching 100%, whereas operation can be done with low mortality and good results.

I must remind you that the essential fault is obstruction to the emptying of the left ventricle, and that life itself depends on the integrity of the function of this chamber. If relief of the obstruction of any hollow viscus or duct in the body is an indication for surgery then obstruction of the left ventricle is surely a paramount indication! Unfortunately, until relatively recently the clinician was often at a disadvantage in giving advice about the need for operation with any degree of certainty, but today there is little excuse for guessing, for we have methods of investigation which allow us to advise with confidence and accuracy.

I will remind you of the 5 phases into which aortic stenosis may be conveniently divided. They are:

1. No symptoms.
2. An awareness of the heart, e.g. palpitations and some dyspnoea at times.
3. Anginal pains, usually with severe dyspnoea.
4. Features of left ventricular failure as shown by congestive attacks, orthopnoea, angina at rest, syncope, etc.
5. Right-sided heart failure.

There is, of course, a certain amount of overlap in these groups, e.g. a patient may be late in phase 3, tending to spill over into phase 4.

From our experience in correlating these phases with electrocardiographic changes, with left heart catheterization, and with operation findings, we can be fairly dogmatic about the need for operation where the diagnosis of aortic *valvar* disease is certain. Thus in grades 3 and 4, operation is essential and in general no pre-operative left heart catheterization is needed. In grade 5, operation is attended with a very high mortality; most such cases are inoperable unless their failure can first be lessened by medical treatment. It is in grades 1 and 2 that it is usually necessary to apply special methods of investigation to assess the severity of the obstruction.

I must also warn against being too easily deterred from operation by the presence of some aortic regurgitation. If the clinical picture is dominated by regurgitation then operation may not be indicated, but it is much commoner to find cases rejected when the degree of regurgitation is modest and the degree of stenosis is dominant. One should concentrate on the degree of stenosis and tend to ignore the regurgitation whenever possible, certainly not use it as an excuse for avoiding operation.

2. FEATURES OF DIAGNOSIS

The electrocardiogram is invaluable if a strain pattern is shown in the left ventricular leads, but this usually occurs in grades 3, 4 and 5. In grades 1 and 2 there may be only slight changes, and this is especially so in children when, in spite of this, a severe stenosis is already present.

Left ventricular puncture combined with estimation of the cardiac output is invaluable and is now the routine practice in most centres.[2] It is quick, simple and safe and can, if necessary, be repeated on successive occasions. We find in grades 3 and 4, when the left chest leads show a severe strain pattern, that the valve gradient is usually not less than 100 mm. Hg, and this is associated with a low cardiac output. Unless there are unusual features we delay our pressure estimations in these groups until the time of operation. However, we press for their routine employment in groups 1 and 2 and especially in young children.

During 1958, at Guy's Hospital, we had 3 examples of children in whom symptoms were absent and the electrocardiographic changes were slight, and yet there was a pressure gradient across the aortic valve of over 100 mm. Hg. One of these children actually developed angina and syncope 3 months later while awaiting admission for open operation. It is not possible to give intelligent and confident advice in these cases without left heart catheterization.

Valvar and Subvalvar Aortic Stenosis

In all young patients and in patients of any age in whom the valve is not demonstrably calcified we demand actual catheterization of the left ventricle so as to obtain a pressure withdrawal record.[3] In this way we can assess whether the stenosis is valvar or subvalvar. In one patient we have even recognized a supravalvar aortic stenosis, confirmed it by a retrograde aortogram, and then operated on it successfully under bypass.

A congenital subvalvar obstruction has been met with in 16 cases and has been made the subject of a special report.[4] In most of these the diagnosis was made by left ventricular puncture, but occasionally, when the stenosis is very near the valve, differentiation of the exact level is not possible and the true diagnosis can then only be made at open operation. I would refer you to the above article[4] for critical discussion of the various technical features; but I would state that at present I prefer a closed transventricular dilatation for most cases of congenital subvalvar stenosis and consider the pre-operative recognition important because one may then be able to avoid the open operation under bypass which is needed when a non-calcified stenosis exists.

Functional Aortic Subvalvar Stenosis

The estimation of the exact level of the stenosis is also essential in recognizing the important condition of functional aortic subvalvar stenosis. This condition has been the subject of two communications[5,6] and I refer you to these for fuller details. Not only is there still wide ignorance of this disease, but even when acquainted with the account of it many clinicians are frankly sceptical. This is unfortunate, because the severity or urgency of symptoms often indicates need for relief by operation whereas operation cannot give relief and in most cases an ill-advised cardiotomy is fatal. I have now met 7 cases of this disease in quite a short time and must emphasize that its recognition is an important

practical matter in aortic stenosis in adults and *in every case* it must be thought of and deliberately excluded.

The most important single observation is the recognition that the valve is not calcified. In aortic valvar stenosis in patients over the age of 30 there is usually little difficulty in demonstrating calcification radiologically. Unless this can be done then the diagnosis of valvar stenosis must be suspect. Occasionally the valve is not calcified in cases of rheumatic disease but in almost every such case the presence of mitral valve disease supplied the true explanation. In all other cases of isolated aortic stenosis it is essential to *prove* the presence of calcification before the diagnosis of valvar stenosis is accepted. Personally, I do not rely upon screening reports, nor upon negative tomography, but insist that plain radiological confirmation be obtained.

If calcification is not demonstrable then it is folly to proceed to operation without a pressure withdrawal record obtained by left ventricular puncture. In this way a diagnosis can be made in a few moments and was, indeed, so made in all our patients. For one reason or another 4 of these were operated on and 3 died at once. The exception was a man aged 32 in whom a lesion was first noticed when he was aged 16. Exploration was done because a congenital fibrous stenosis could not be excluded. Operation revealed a functional obstruction, and fortunately, he did not die. Other surgeons have encountered such cases in the early twenties; so it must be thought of and if possible excluded, even at this young age.

In one of my patients the predisposing factor was left ventricular hypertrophy from previous systemic hypertension. This was not a factor in the remainder, in whom we have to fall back upon the explanation of an obscure cardiomyopathy. I do urge you not to ignore the great importance of this new disease of the left ventricle—functional aortic subvalvar stenosis—hitherto unrecognized until revealed by left ventricular pressure withdrawal records.

3. TYPES OF OPERATION

Much of the time that is spent arguing about the exact type of operation to be done for aortic stenosis could be better spent in emphasizing the basic need for an operation of any sort. More patients are dying or continuing unrelieved of their disability without any operation than are being submitted to open or closed procedures. I have visited clinics and hospitals in many parts of the world and am impressed with the continuing reluctance to advise and to use surgery in this serious disease. Even when operation is used it is commonly only for the advanced or desperate cases, rarely at the proper time when there is the greatest chance of a good result with a low mortality.

Non-calcified Aortic Valvar Stenosis

I have already mentioned that I prefer a closed transventricular operation, but that an open operation is essential for a non-calcified valvar stenosis. Those of us who have tried closed dilatation on such cases know that the smooth, high, dome-shaped valve is not amenable to safe splitting with an instrument. The valve cone is torn and damaged so that severe regurgitation follows. The result is either fatal or an aggravation of the clinical state. Visual division of the fibrous valve cone is essential.

In common with many surgeons I have done this under hypothermia and have in fact operated on 10 patients in this way. Three patients died and the result was good in 5 and not so satisfactory in 2, in whom substantial regurgitation occurred. It is now my custom to use total heart-lung bypass for these cases because the longer time available is desirable for 3 reasons. First, one can do the actual valvotomy with greater prevision when unhurried; second, one can spend adequate time on careful suturing of the aortic wall incision; third, the ability to give powerful coronary perfusion is invaluable in avoiding or correcting arrest of ventricular fibrillation when the muscle is in poor condition. I find, in fact, that I use some 30 minutes of bypass for these cases and would hate to go back to the hurried work needed to complete the delicate task in the 10 minutes available with hypothermia. The safe and secure suturing of the aortic wall is especially important. It may be possible to rely upon a lateral clamp and then to complete the suture at leisure, but this may fail and then disaster can follow.

The avoidance of regurgitation is paramount, and when a homogeneous, smooth dome is displayed (as is the case in severe stenosis) I prefer to make only 2 incisions, thus producing a bicuspid valve. However, in certain cases, and especially the less severe ones, the 3 commissures must be cut and then the greatest care must be taken to cut exactly in the line of the fused edges. It is so easy to transgress to one or other side, and regurgitation then follows. This is much more prevalent and much more significant than after open pulmonary valvotomy, presumably because of the higher systemic pressure to which the valve is exposed. It can be a great disappointment when substantial regurgitation follows an open valvotomy, and this is scarcely mitigated by the hope that as the valve scleroses in later years the regurgitation may lessen.

I have not used any form of artificial coronary perfusion, either forward or retrograde; nor have I used potassium arrest in these cases. I have simply clamped the aorta and anoxic arrest has followed in all the 6 cases in which I have used bypass, but there was no difficulty in any of them in starting normal action as soon as the aortic clamp was removed and normal coronary perfusion restored. Unless or until I encounter difficulty with this I do not intend to enter upon the complications of artificial coronary perfusion for periods of half an hour. For longer operations there may be more need for it.

There has been one post-operative death in 6 open cases under bypass.

I have also stated that in some cases of congenital subvalvar stenosis a pre-operative pressure withdrawal record may fail to exclude a valvar stenosis. Also in some severely ill patients we have decided against a pre-operative withdrawal record. Actually today, with our greater experience of this technique, it is doubtful if the examination should ever be omitted. If, however, doubt remains and it is felt that an open operation under bypass, with all that it implies, is to be avoided if possible, a simple policy can be used. For such cases I make a smaller anterior incision along the line of the left sixth rib and expose the left ventricle low down so that a catheter can be introduced easily at the apex to give a straight run along the line of the axis of the outflow tract for a pressure withdrawal record. If this now shows a subvalvar stenosis I insert an expanding dilator and split the fibrous stricture

open. This quick operation is well tolerated and sometimes gives an excellent result with substantial obliteration of the gradient, I am, as yet, unconvinced that a great deal more can be achieved by means of an open operation from above. Access is so restricted that a full and adequate resection of the subvalvar stricture is very difficult. Although surgeons state they can do this, the evidence is as yet slight and the reports in the literature of actual pressure changes are scanty and include no large series. We must await further information before we can decide this point and in the meantime I intend to continue using transventricular dilatation except when a subvalvar stenosis is unwittingly exposed from above.

I must point out that in these cases an element of functional obstruction secondary to left ventricular hypertrophy is a superadded phenomenon, just as occurs in the right ventricle in many cases of pulmonary stenosis. In fact in severe cases it may be impossible to relieve the stenosis substantially either by an open or by a closed procedure. In this event we must await the natural process of recession of the muscular hypertrophy, by which means the high intraventricular pressure will gradually drop to a normal or near-normal figure. It is quite certain that this can occur in the left ventricle for it has been observed and recorded, although as yet not so often as in the right ventricle.

I would mention that congenital subvalvar stenosis is, in my experience, usually a very severe condition and I always think of it as being likely to be present when a young patient has a very severe stenosis.

Calcific Aortic Valvar Stenosis

When we come to calcific aortic valvar stenosis the problem of the best type of operation is more complex. In patients under the age of 45-50 years there is obviously a good case for an open operation, especially when the valve is apparently not heavily calcified. I think one must accept the desirability of an open operation in these patients.

In practice I have found that so many patients in the thirties or forties have such severe features of the disease when they come for operation, often having been on the verge of left ventricular failure, that one fears the greater burden of an open operation and still turns to a quick closed transventricular dilatation, which has given such a high proportion of good results.

Open operation under hypothermia now holds no attractions for me in these patients, I have used it in 6 cases with 3 survivals. The short time at one's disposal limits the time available for safe suture of the aortic incision, and I consider this an important danger in these cases. Even more important is the danger of irreversible ventricular fibrillation, and this was the usual mode of death in my 3 fatal cases. The coronary perfusion provided by the pump oxygenator seems to be the best means of supporting the strained left ventricle.

For the same reasons I am unattracted by the quick in-and-out open operation done under fluothane anaesthesia, which gives even less time than hypothermia.

For patients in the fifties and early sixties (operation is rarely indicated later than the early sixties) I am unattracted by open operation under bypass and unhesitatingly perform a closed transventricular dilatation. This procedure carries a low mortality, as is shown by the fact that in my last consecutive 100 cases there were only 7 deaths. The results are good in some 70% and in many the improvement is dramatic. They include many patients in the most advanced stages of aortic stenosis. The number of relapses in which a second valvotomy is needed has been small, some 5 or 6 patients only. I have in fact done a second operation in only one patient, a man aged 35, and he succumbed to an open procedure under hypothermia.

The incidence of regurgitation, either inflicted or aggravated, has been small, doubtless due to the rigidity of the calcified valve structure. In passing, one must decry the fears so often expressed that heavy calcification is a contra-indication to operation. This is just not so, for many very good results have been obtained in its presence. As a balance to the few cases which have suffered some regurgitation, there are those in whom pre-operative regurgitation has been lessened or virtually obliterated as a result of the improved mobility of the cusps secured.

I hold no brief at all for the various closed transaortic procedures by which instrumental or finger dilatation is used blindly from above. I am totally unconvinced of their supposed advantages and am unattracted by the extra technical hazards involved. The transventricular operation gained disrepute when it was done with a large, clumsy and dangerous type of instrument. If a simple two-bladed dilator is used, only the thickness of an ordinary lead pencil, it causes no damage to the ventricle and no controlling purse-string stitch is needed. It can be slipped in easily and the whole procedure takes barely 60 seconds. During this short time the brain is protected from calcific emboli by temporary occlusion of the head and neck aortic branches. Simple finger pressure controls the small incision while 2 or 3 stitches are inserted.

While continuing to use this simple, quick procedure for most cases I am now gradually building up my own experience with open valvotomy under bypass. Clearly it is essential to prosecute this open method as much as possible until we know how much extra risk it carries. At present the evidence that exists seems to show that it in fact carries a substantial extra mortality. It is difficult to secure precise figures in long enough series to form a final opinion. It would seem that in the best hands the open operation under bypass carries a mortality of some 25% and in average hands the mortality is about twice this.

It is inevitable that one is influenced by this high risk when faced with the individual patient and when one knows that a simple closed operation carries a risk of less than 10%. This would seem to compel caution, at present, in selecting cases for the more severe, even if more exact, open procedure.

It will be observed from my composite figures, given above, of open operations under hypothermia and under bypass, that the total is 22 with 7 deaths; these include both calcified and non-calcified cases.

SUMMARY

The subject is treated under the 3 headings of (1) indications for operation, (2) diagnostic features, and (3) types of operation.

Under (1) stress is laid on the danger of withholding or delaying operation in suitable cases, and on the fact that in such cases the mortality rate is far higher if operation is withheld.

Under (2) reference is made to the diagnostic value of ECG and of pressure gradient. Left ventricular puncture combined with estimation of cardiac output is invaluable. The importance of the differentiation of valvar from subvalvar stenosis is mentioned, and particularly of the newly recognized 'functional aortic subvalvar stenosis'. The diagnosis of the latter condition is discussed and the danger of cardiotomy if it is present. Stress is laid on the importance of demonstrating calcification in excluding this condition.

Under (3) the place of the closed transventricular operation and of the open operation is discussed, and the value of the heart-lung bypass in aortic stenosis. The open operation is preferred in non-calcified aortic valvar stenosis, but in the calcific condition the problem of the best type of operation is more complex and the author discusses the relative merits of the different available procedures in various types of case.

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RESISTANCE OF THE BODY LOUSE (*PEDICULUS HUMANUS CORPORIS* DE G.) TO DDT POWDERS*

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Reports from numerous countries throughout the world have indicated that DDT powders are apparently no longer giving adequate control of the body louse. This suggests that this insect pest has possibly developed resistance to DDT, just like the house fly, which was previously adequately controlled by this insecticide. As resistance is a serious public-health problem, the World Health Organization's Division of Environmental Sanitation has arranged body-lice resistance tests with standardized kit forwarded from Geneva. The present investigations were conducted at Queenstown, Cape Province, and neighbouring areas as part of the global survey.

* Published with permission of the Secretary for Health, Pretoria.

In 1954 and 1958 the powders from the standard WHO kit for determining the resistance of the body louse were used on cloth according to the kit instructions.

In 1956, at Queenstown, Dr. Carroll N. Smith, Entomology Research Division, US Department of Agriculture, Florida, confirmed our 1954 results. In addition to the WHO powder test kit, he used cloth pads impregnated with acetone solutions of insecticides for beaker tests as described by Eddy.[1]

Lice were tested from the Bantu locations not only of Queenstown but also of Eskeaton, Macibini, Qoqodala and Mtyantya, in the Queenstown area. This was done in order to obtain a clearer insight into the local problem, since less DDT had been used in these neighbouring locations than in the Queenstown location. The average percentage resistance of lice from individual areas is shown in Table I. Only lice

TABLE I. AVERAGE PERCENTAGE RESISTANCE TO INSECTICIDE POWDERS

Concentration of toxicant in powder	*1954*				*1956*	*1958*		
	Queenstown		*Eskeaton*	*Macibini*	*Queens-town*	*Qoqodala*	*Matyan-tya*	*Queens-town*
	Series 1	*Series 3*	*Series 2*	*Series 4*	*Series 1*	*Series 1*	*Series 2*	*Series 3*
0·04% DDT	—	—	—	—	81·7	43·3	39·8	—
0·1% DDT	67·5	60·0	54·2	35·0	—	—	—	33·3
0·2% DDT	—	—	—	—	71·7	36·7	43·3	—
0·5% DDT	52·5	12·5	49·4	2·5	—	—	—	40·0
1·0% DDT	71·7	38·8	76·7	12·5	75·0	6·7	10·0	33·3
5·0% DDT	81·7	28·8	56·7	5·0	43·3	0	0	25·0
0·02% gamma BHC	—	—	—	—	46·7	13·3	0	—
0·1% gamma BHC	—	—	—	—	26·7	—	—	—
0·25% gamma BHC	0	0	0	0	—	—	—	0
0·5% gamma BHC	0	0	0	0	0	—	—	0
2·5% gamma BHC	—	—	—	—	0	0	0	—
0·0016% pyrethrins	—	—	—	—	21·7	0	6·7	—
0·008% pyrethrins	—	—	—	—	8·3	—	—	—
0·02% pyrethrins	0	0	0	0	—	—	—	0
0·04% pyrethrins	0	0	0	0	0	—	—	0
0·2% pyrethrins	—	—	—	—	0	0	0	—
Controls normal	95·8%	100%	100%	100%	100%	93·3%	90·0%	100%
Average temperature	17·3°C	25·1°C	16·0°C	25·1°C	24·7°C	26·9°C	25·5°C	26·4°C
Average rel. humidity	—	—	—	—	—	38·5%	42·4%	40·0%

which appeared normal after 24 hours' exposure, were scored as resistant. Since 1954 some of the concentrations of insecticides provided in the test kit have been altered or added; hence the blanks in Table I. Replicates were run as specified in the kit instructions and the average percentage resistance calculated.

COMMENTS

Queenstown Location

From June 1952 until November 1956 10% DDT in talc was regularly used at intervals of 6 weeks for dusting premises, persons and clothing against louse-borne typhus in this Bantu location. After November 1956 0·6% lindane was employed selectively on persons and clothing found infested.

1954. For series 1 (1954) lice were obtained from an old invalid Bantu woman who continued to be infested in spite of repeated deverminization between the regular 6-weekly dustings. Her lice were found to be 81·7% resistant to 5% DDT powder. (It is of interest to mention that the Bantu woman who always tended this invalid was carefully searched and no lice were found on her person or clothing, although she always slept in the same room as the invalid. All occupants slept on the floor.)

For series 3 (1954) lice were obtained from a Bantu male. These were less resistant than the lice in Series 1 (1954).

Commenting on the 1954 tests the following statement was published in the *Chronicle of the World Health Organization*:[2] 'Two samples from the African township of Queenstown and Eskeaton, Union of South Africa, with mortalities of 18% and 14% respectively, were the most resistant strains encountered in the survey, even surpassing those from Korea in this respect.'

1956. Commenting on series 1 (1956) Smith[3] stated: 'The dust tests indicated a high resistance to DDT, and the beaker tests showed the degree of resistance to be in the neighbourhood of 130 times normal. There was little or no resistance to lindane . . ."

1958. At Queenstown location in July-August 1958. Kit prepared in February 1954 had to be used for series 3 (1958). (Fortunately kit prepared in June 1957 was available for the 1958 tests at Qoqodala and Matyantya.)

Resistance to DDT as shown by series 3 (1958) was consistently lower than the Queenstown values during 1954 and 1956 (except for the 0·5% DDT figure in series 3 (1954). Resistance to 1·0% DDT was respectively 5 times and 3·3 times as high as in Qoqodala and Matyantya. Resistance to 5·0% DDT was 25·0%.

Eskeaton Location, 1954

Since the Bantu came to know of the usefulness of DDT as an insecticide, which was about 1948, they have been buying it in small quantities from traders for private use. It has not, however, been used on a large scale.

From 1949 to 1954 only sporadic typhus outbreaks occurred in this area. DDT dusting by the Government Health Department was confined only to spots where outbreaks actually occurred. All contacts were traced and dusted to quell the outbreaks and to stop further spread of the disease. Except for these outbreaks no large-scale control measures were carried out.

The very high resistance values found at Eskeaton in series 2 (1954) are of interest especially because relatively little DDT had been used in Eskeaton location as compared to the Queenstown location.

Macibini Location, 1954

This area is not densely populated and huts are scattered. This location is in the Glen Grey district, which was an endemic plague area, and dusting campaigns with 10% DDT had been carried out by the Government Health Department throughout this district to suppress plague outbreaks. The last campaign was conducted in 1949. Since then some of the Bantu have been buying DDT themselves, but some kraals have not yet used this insecticide.

Table I shows that in 1954 DDT resistance in this location was markedly lower than at Queenstown and Eskeaton locations.

Qoqodala Location 1958

DDT has not been used for lice control in this location. It is thus instructive that Table I shows a markedly and consistently lower DDT resistance in 1958 at Qoqodala than that found in the DDT-controlled Queenstown location during 1954 and 1956. Of special interest is the complete absence of resistance to 5·0% DDT.

The resistance to 0·02% gamma BHC was 13·3%.

Matyantya Location, 1958

As at Qoqodala, DDT has not been used for lice control in Matyantya location, and Table I discloses in Matyantya, as in Qoqodala, a markedly and consistently lower DDT resistance than in the DDT-controlled Queenstown location during 1954 and 1956.

There was 10·0% resistance to 1·0% DDT, no resistance to 5·0% DDT, none to 0·02% gamma BHC, and 6·7% resistance to 0·0016% pyrethrins.

EFFICIENT DUSTING

In September 1954, when the highest DDT resistance was found in Queenstown lice from the old Bantu invalid who remained infested in spite of repeated deverminization between the regular 6-weekly dusting, we reported: 'After collection of test lice material . . . , the premises, consisting of 2 rooms, together with all bedding and clothing and 3 occupants, were thoroughly dusted with 10% DDT in talc by members of the Field Staff of the Government Health Department. *After 24 hours all lice were dead* except 2 paralysed ones. Eight subsequent daily observations revealed no live lice on the premises.'

This finding shows that in 1954 the Queenstown lice, which had the highest DDT resistance of all lice tested with WHO kit in about 100 parts of the world, were 100% controlled and completely killed by proper and efficient application of 10% DDT powder.

We further stated: 'Field Assistant J. G. Greeff reported that "people who were deverminized with 10% DDT powder on 15 July 1958 were again searched for lice on 17 July 1958, but not a single louse was seen". This information refers to that part of Qoqodala location in which DDT was previously used, and shows that if properly applied, DDT still gives a 100% kill.'

SUGGESTIONS FOR FUTURE TESTS

Experimental Dusting to Support Test-kit Data

As the South African lice were the most resistant of all lice tested with WHO kit in about 100 different parts of

the world, and as we still obtained a 100% kill by proper application of DDT powder, we would like to suggest that future tests with WHO kit might consistently be supplemented by thorough experimental dusting of 2 or 3 premises and the occupants as well as their clothing. This will not only yield information on resistance, but will also show the relative efficacy of DDT when properly applied.

Every experienced entomologist knows that what happens in laboratory tests is not always repeated in the field. At Letaba,[4] where we doubled the commercial value of the citrus crop in certain experimental plots by keeping ants out of the trees, we found a 100% kill of red scale on oranges which were sprayed with DDT + Avon oil; but when a few trees were similarly sprayed, red scale reproduced so prolifically that the trees defoliated because natural scale enemies were exterminated.

Gravidity Data to Supplement Busvine Tests

Following the Queenstown and Letaba considerations, we have further developed a simplified gravidity technique for detecting physiological resistance of *Anopheles gambiae* Giles to BHC in the field during malaria eradication campaigns,[5] because resistance results determined in a laboratory test might be different from resistance under field conditions which might affect eradication procedures.

Resistance of Immature Lice

It might be of practical value to obtain information on the relative resistance of immature and mature body lice, since a more complete insight into resistance will effect better control.

SUMMARY

1. The resistance of body lice was determined with the WHO test kit at Queenstown and neighbouring areas.

2. In September 1954 resistance to 5·0% DDT powder at the Queenstown location, where DDT had been regularly used at 6-weekly intervals for typhus control since June 1952, Eskeaton location, where less DDT had been used, and Macibini location, where still less DDT had been used, was respectively 81·7, 56·7 and 5·0%.

3. In November 1956 Dr. C. N. Smith, of Florida, USA, confirmed our 1954 data and found that DDT resistance was in the neighbourhood of 130 times the normal.

4. In August 1958 in Qoqodala location, where DDT had not been used for lice control, resistance to 1·0% DDT, 5% DDT and 0·02% gamma BHC was respectively 6·7%, 0% and 13·3%. At Matyantya location, where DDT had similarly not been used, resistance to 1% DDT, 5% DDT, 0·02% gamma BHC and 0·0016% pyrethrins was respectively 10·0%, 0%, 0% and 6·7%.

5. As the Queenstown lice were the most resistant of all lice tested, but were still 100% killed by proper application of 10% DDT, it is suggested that experimental dusting of 2 or 3 premises as well as the occupants and their clothing should be carried out to supplement future tests with the WHO test kit.

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A CLINICAL TRIAL OF MELLERIL (TP-21) IN THE TREATMENT OF MENTAL DISORDERS

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A new phenothiazine compound, 3-methylmercapto-10-{2′ [N-methyl-piperidyl-(2″)]-ethyl-(1′)}-phenothiazine, was administered to 14 chronic, deteriorated male European schizophrenic patients and 1 paraphrenic patient. The ages of the patients varied from 30 to 40 years, and the duration of their illness from 5 to 22 years. Of these patients 14 had previously received electric convulsive therapy (ECT) and some had received insulin coma therapy or had been treated with reserpine or chlorpromazine. Three patients had undergone prefrontal bilateral leucotomies approximately 10 years previously.

Details concerning age, duration of illness, diagnosis, dosage of Melleril, symptoms before treatment with Melleril, effects of treatment, side-effects, other forms of treatment given previously, comparison with other forms of treatment, and duration of treatment are listed in Table I.

CLINICAL RESULTS

The assessment of the results of the trial was made on clinical impressions of a qualitative nature and no additional control cases were included. However, in view of the long duration of their illnesses and the different kinds of treatment given to them, these cases could, to some extent, serve as their own controls.

Of the 15 cases treated with Melleril, 6 (40%) showed a moderate to marked improvement. Of the 14 who had received ECT previously, 5 responded better to treatment with Melleril than to ECT. Of the 4 cases who had received insulin coma therapy, 2 responded better to treatment with Melleril than to insulin therapy. In 5 cases chlorpromazine, reserpine and Melleril were used. In 3 of these Melleril was equally effective to the other 2 drugs, and in 2 Melleril led to a marked improvement. Of the 3 leucotomized patients to whom Melleril was administered, 1 showed a marked improvement.

In no case did Melleril lead to an exacerbation of the patient's mental condition. No patient reached the level of complete remission; but the chronicity of the illness and the prolonged period of institutionalization of the patients in the trial should be kept in mind in this connection.

DISCUSSION

Melleril has both tranquillizing and antipsychotic properties. Its activating properties appear to be low compared with trifluoperazine, and of the same order as chlorpromazine. The sedative action of the drug is less marked than that of chlorpromazine. There is no specific relationship between dosage, length of treatment, clinical effects, and degree of relapse after cessation of treatment.

The therapeutic effect of the drug appears to be symptomatic only; 2 cases relapsed completely on cessation of administration of the drug.

The 2 hebephrenic patients both failed to respond to treatment; both were, however, chronic cases (duration of illness 14 and 15 years respectively) and both patients had had leucotomy operations.

No extrapyramidal symptoms occurred during the trial and the clinical improvement was not dependent on the occurrence of such symptoms.

Owing to the relative absence of side-effects, the patients remained amenable to group psychotherapy and group discussion. On the whole the trial had a beneficial effect on the patients.

Side-effects. One chronic catatonic patient, aged 48 years, suffered from an increased incidence of asthmatic attacks after 37 days of administration of the drug (25 mg. *t.d.s.*). It is possible, however, that this may have been a coincidental finding. The mental status of this patient remained unchanged by Melleril.

TABLE I. SUMMARY OF CLINICAL FINDINGS

Case	*Age (years)*	*Duration of illness (years)*	*Diagnosis*	*Dosage*	*Symptoms before treatment with TP-21*	*Results of treatment with TP-21*	*Side-effects*	*Other kinds of treatment previously given*	*Comparison with other kinds of treatment*	*Duration of treatment*
1	39	15	Schizophrenia, hebephrenic	50 mg. *t.i.d.*	Withdrawn, religiose, vague persecutory delusions	No change	Nil	Electric convulsive therapy (no lasting effects), prefrontal leucotomy (no lasting effect)	As ineffective as previous treatment	21 days
2	45	22	Catatonic	400 mg. *t.i.d.*	Withdrawn, severe autism, emotional blunting, stereotypy of gait, mannerisms	No change	Nil	Electric convulsive therapy (no lasting effect)	As ineffective as ECT	33 days
3	48	15	Catatonic	25 mg. *t.i.d.*	Severe withdrawal and autism, out of contact with reality, ideational poverty	Mental status unchanged (asthmatic attacks)	Increased incidence of asthmatic attacks	Electric convulsive therapy (no lasting effects)	Mental status unchanged (asthmatic attacks)	37 days
4	43	20	Catatonic	250 mg. *t.i.d.*	Asocial, withdrawn, incoherent, incontinent	Fairly sociable, continent, answers readily, fair initiative	Nil	Electric convulsive therapy (no lasting effects)	More effective than ECT	6 months
5	30	10	Catatonic	150 mg. *t.i.d.*	Asocial, constantly escaping, irrelevant, slovenly, indolent	Cooperative, neat, replies readily, no longer escapes, became a leader and organizes soccer games	Somnolence, countered by methylphenidyl acetate	Electric convulsive therapy (no effect)	Far more effective than ECT	7 months
6	32	7	Catatonic	100 mg. *t.i.d.*	Mutistic, incontinent, immobile with cyanosis, sores on legs	Replies rationally and to the point, no cyanosis of legs, no sores, plays cricket, works in garden, continent	Nil	Electric convulsive therapy (no effect)	Far more effective than ECT	3 months
7	30	10	Catatonic	50 mg. *t.i.d.*	Aggressive, mutistic, slovenly, destructive	Fair replies, neater, no longer destructive, on ground parole	Somnolence, countered by methyl phenidyl acetate	Electric convulsive therapy and insulin coma therapy, chlorpromazine, reserpine	Superior to electrical and insulin therapies; chlorpromazine and reserpine had no beneficial effect	3 months*
8	38	15	Paranoid	500 mg. *t.i.d.*	Deluded, disorientated for self and surroundings, irrelevant, grandiose	Poor response but patient gives less expression to delusional system	Nil	Electric convulsive therapy and insulin coma therapy	Equal to electric convulsive therapy, better than insulin coma therapy	5 months†
9	42	17	Schizophrenia, catatonic	400 mg. *t.i.d.*	Mutistic, aggressive, destructive out of contact with reality, filthy, hyperactive, incontinent	Not destructive, tidy, not hyperactive, continent, rational, but gives a limited account of himself A degree of insight present	Nil	Bilateral standard leucotomy, electric convulsive therapy, insulin coma therapy, reserpine chlorpromazine (poor effect)	Superior to other kinds of therapy	6 months
10	40	12	Catatonic	300 mg. *t.i.d.*	Aggressive, withdrawn, gave very poor account of self	Answers more freely but remains dull and retarded, not aggressive	Nil	Bilateral standard leucotomy, electric convulsive therapy (no effect)	Superior (?) to other kinds of therapy	5 months
11	40	14	Hebephrenic	350 mg. *t.i.d.*	Manneristic, silly, fatuous, retarded and irrelevant, hoards rubbish	Slightly less manneristic, but still hoards rubbish, retarded, irrelevant, fatuous	Nil	Bilateral standard leucotomy, electric convulsive therapy, chlorpromazine, reserpine	Equally ineffective	7 months
12	34	9	Catatonic	300 mg. *t.i.d.*	Stutters, manneristic, nonsensical, incontinent	Less frequently incontinent, otherwise unchanged	Nil	Electric convulsive therapy, insulin coma therapy, chlorpromazine, reserpine	Equally ineffective	3½ months
13	54	12	Paranoid	25 mg. *t.i.d.*	Delusions of persecution in respect of wife and former employer	Delusions controlled but patient developed no real insight	Nil	Chlorpromazine, reserpine, electric convulsive therapy	Equally effective	3 months

TABLE I. SUMMARY OF CLINICAL FINDINGS

Case	Age (years)	Duration of illness (years)	Diagnosis	Dosage	Symptoms Before treatment with TP-21	Results of treatment with TP-21	Side-effects	Other kinds of treatment previously given	Comparison with other kinds of treatment	Duration of treatment
14	45	17	Catatonic	200 mg. *t.i.d.*	Short tempered, severely withdrawn, manneristic, gives poor account. Delusions of persecution	More cooperative, no longer aggressive, no more delusions	Nil	Prefrontal leucotomy	More effective than prefrontal leucotomy	3 months
15	51	5	Paraphrenic	300 mg. *t.i.d.*	Hyperactive, deluded, hallucinations, disorientated, disturbed sequence of thoughts	More composed, interested in gardening, volunteers for work, not impulsive yet still asocial, not hallucinated, not deluded	Nil	Electric convulsive therapy (no lasting effect)	Superior to ECT	5 months

* Patient relapsed completely when tablets were unavailable.
† Delusions became more florid when tablets were unavailable.

No psychological or neurological side-effects were noted and there were no side-effects relating to the cardiovascular or gastro-intestinal systems or to the skin and mucous membranes. There was no alteration in the weight of the patients.

Somnolence was fairly marked in 2 cases, but this was readily countered by moderate doses (20-40 mg. per day) of methylphenidylacetate. No blood dyscrasias were observed.

Dosage. The dosage of Melleril varied from 25 mg. 3 times daily to 500 mg. 3 times daily.

Conclusion. It appears that Melleril is non-toxic, fairly free from side-effects, and has a wide dosage range. It does not interfere with normal ambulatory and psychomotor activity and does not impede psychotherapy.

SUMMARY

1. This study concerns the treatment of 15 chronic schizophrenic male European patients with Melleril.

2. In no instance was the therapeutic effect of Melleril inferior to that of other kinds of therapy given previously. The effect of Melleril was equal to or slightly superior to that achieved with other methods of treatment in 8 cases (53%), and moderately or markedly superior in 7 cases (47%).

Throughout the trial the patients remained amenable to individual and group psychotherapy.

3. The nature of the improvement in these chronic cases seems to be symptomatic rather than curative, and no patient reached the level of complete remission.

4. The only side-effect noted was somnolence; however it was less marked than with chlorpromazine. This side-effect was easily controlled by the administration of methylphenidylacetate. Attacks of asthma occurred more frequently during the trial in 1 asthmatic patient—it is possible that this exacerbation of the asthma may have been caused by Melleril. The activating properties of Melleril were found to be low, approximately equal to those of chlorpromazine.

5. Improvement of the patients' mental status was in no way associated with the occurrence of involvement of the extrapyramidal tract.

I am indebted to Messrs. Sandoz Pharmaceutical Products for supplies of Melleril which were used in this work.

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DRAINAGE TUBES IN UROLOGY*

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My attention was first drawn to the importance of the accurate placing of drainage tubes long before I confined myself to urology, at a time when I was doing a fair amount of gall-bladder surgery. I did not always drain the gall-bladder area after cholecystectomy, but when there was a likelihood of the oozing of blood or bile I found that if I stitched my drain lightly to a non-vascular fold of peritoneum in close proximity to the area I wished to drain, the patient convalesced better and quicker than if I merely 'popped in' a tube. I am sure that a tube just 'popped in' wanders away from the vital area it is supposed to drain owing to respiratory movements, coughing, tossing around, bowel movements, etc.; and the result is that the whole peritoneal cavity must first of all fill up with blood and bile before these fluids find exit along the drainage tube, and there is increased tendency to ileus, peritonitis, pain, and residual abscess formation.

A drain was 'popped in' at an operation I witnessed for removal of a stone from a ureter at the pelvic brim. The patient leaked urine here for weeks, even after the ureter was catheterized transvesically. Eventually extensive scar tissue formed, the ureter became stenosed and, when this was explored and the ureter mobilized, the common iliac vein was so adherent to the ureter that it was torn into with what might have been serious consequences to the leg. The kidney was so damaged that in the end a nephrectomy had to be done.

The *pathological process* is as follows: The drain that has been 'popped in' moves freely in the extensively dissected cavity, so that urine fills the cavity before it finds the tract alongside the tube. The ureter had already had its blood supply interfered with by dissection, and now it is surrounded by urine with its necrotizing effect. This leads to devitalization, a big cavity which turns septic, and extensive scar formation around a severely damaged ureter. What can we expect but extensive stricture formation with all its accompanying risks and complications?

To avoid this, use corrugated tubing two or three gutters wide (or other rubber tubing) with the end cut smoothly round. Anchor this end in close proximity to the area you wish to drain, by means of thin plain catgut. If one has done a uretero-lithotomy the stitch is put through the fascia over the psoas muscle, for example, and the end of the tube is so placed that it does not actually rest on the stitches placed in the ureteric wall.

I almost always bring the drain through a stab wound and not through the main wound. This has the advantage (*a*) that you

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can stitch the main wound more securely, and (*b*) that if you are draining a septic area the main wound does not become septic. If you have reason to fear that it might become septic, place in it small separate drains, draining only the layers of the abdominal wall. This avoids prolonged wound sepsis, breaking down of the wound, and the development of hernia.

One point about stab drains: There is no need to force your knife right through the whole thickness of the abdominal wall. By so doing you run a risk of stabbing a large vessel. Merely cut through the skin, force a large artery forceps through the other layers, and open it widely; then grasp the tube and bring it out.

If there is a near-by stitch, put the safety pin under the stitch and through the tube. If there is no stitch, make a hole through the skin in order to anchor the tube, which should be secured, not only against slipping inside, but also against coming out when the nurse struggles to get hard caked dressings off the wound. When a tube is removed too early there is not yet an adequate tract through which urine, pus and blood can find their way out, and abscess cavities form inside muscle layers that have closed over, with consequent pyrexia, destruction of tissues, etc.

When the tube is shortened, pull the pin just far enough out to release the tube, but leave the pin through the skin. It only hurts to pull it right out and then to push it in again through the same hole. The thin plain catgut by which the tube is anchored will allow the tube to be partially withdrawn 3 or 4 days after its insertion, by means of a gentle tug. Urine, etc. find their way along the tube without first having to spread far and wide, and when 3 or 4 days later one shortens the tube by half an inch, tissues fall in behind the tube, thus closing the fistula. By this time a fairly good tract has formed and surrounding tissues have adhered to one another beyond the actual leaking area; thus the main danger of big cavity formation has been avoided. It now only remains to shorten the tube judiciously day by day, according to the amount of drainage. If the amount is gross, then do not shorten; if little or none, shorten fairly rapidly.

My experience in a considerable number of cases so drained has been that there has been slight or fair drainage for the first 24-48 hours, and then the tract closed spontaneously, sometimes soon after the first shortening, sometimes even before the tube is shortened at all.

Types of Operation

This method of drainage has been found advantageous in the following operations:

(*a*) *Uretero-lithotomy.* This is probably the most important indication for attending to the minute details that have been mentioned.

(*b*) *Renal pelvis operation.* In this it is my practice, after having repaired the pyelolithotomy incision, to stitch the corrugated drain to a thin piece of tissue just lateral to the suture line, so that the drain never lies on the stitches and over the wound in the pelvis.

For large hydronephrotic pelvises I have done the Heyns-Anderson pyeloplasty with good results and, even in babies of a few months old, it is usually not necessary to splint the pelviureteric anastomotic site with an indwelling catheter; but, especially in children with thin ureters, where oedema after the operation could easily block the pelvic outlet and so cause a break-through somewhere else in the newly constructed pelvis, I deliberately cut a window in the posterior wall of the pelvis, and to the edge of this window I secure a corrugated drain. Urine leaks out here freely for 2-3 days, and yet, because the drain is not in the kidney pelvis or ureter, these cavities do not become infected. (On the same principle one incises a septic joint and then secures the drain to the capsule, but not *into* the capsule.) After 3 days the drain is shortened by ½-1 inch. By this time there is a tract along which urine can reach the drain, but if the pelvi-ureteric junction is adequately patent, tissues quickly fall in and shut the 'window' off. Some of these cases do not leak urine at all, or perhaps only for 24 hours. In such cases one could perhaps have dispensed with the window and the drain, but I have never found them do harm or delay healing and convalescence in any way.

(*c*) *Uretero-sigmoidostomies and uretero-ileostomies.* In these I similarly drain the anastomotic sites. Even if there is no urinary leakage, a fair amount of serum always comes out past the drains, and it is comforting to know that urine or bowel content will have ready exit should anything go wrong with one's suture lines.

(*d*) When *draining the retropubic or prevesical space* one is draining a cavity with rigid walls. Here there is no need to secure the drain with stitches, but it is nevertheless important to place it accurately, and to secure it to the skin in some way; for I am sure that if this drain is removed too soon, by accident or on purpose, then there is a real danger of retropubic abscesses and osteitis pubis.

(*e*) *After removing large vesical diverticuli* it is again an advantage to stitch the drain near where the neck of the diverticulum was cut through, because leakage not infrequently takes place at this site.

SUMMARY

Reasons are given why accurate drainage should be instituted after the opening of urine-carrying hollow organs.

Details are given of how this can be achieved.

A few operations are quoted where such drainage has been instituted with satisfying results.

TRANSVAAL SOCIETY OF PATHOLOGISTS

SUMMARIES OF SCIENTIFIC PAPERS*

THE a-z-z_{35} GROUP OF SALMONELLA ANTIGENS

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The serological relationships of the salmonella flagellar antigens *a*, *z* and z_{35} have been clarified by means of a series of agglutinin-absorption tests. The results show that although there is no direct relationship between *a* and *z*, they each have a fraction in common with z_{35}. Furthermore, there appear to be two varieties of the *z* antigen, as exemplified in *S. poona* and *S. bulawayo*.

OSSIFYING FIBROMAS AND THEIR RELATION TO FIBROUS DYSPLASIA AND OTHER TUMOURS: A HISTOCHEMICAL STUDY

DR. W. J. PEPLER, *Institute for Pathology, Pretoria*

The alkaline and acid phosphatase distribution was studied in 4 cases of ossifying fibroma and in 1 case of fibrous dysplasia. It was demonstrated that the so-called fibroblast in ossifying fibroma is functionally an osteoblast, and it is therefore proposed that these tumours be regarded as benign osteoblastomas. The relationship between fibrous dysplasia, ossifying fibroma, osteoma, and osteoclastoma was briefly discussed.

*Read at a meeting of the Society, held in Johannesburg on 17 October 1959.

THE INCIDENCE OF DIABETES MELLITUS IN ONE DISTRICT OF BASUTOLAND

DR. W. M. POLITZER, *South African Institute for Medical Research, Johannesburg, and* DRS. B. HARDEGGER *and* T. SCHNEIDER

In this investigation of 3,000 Basutos in the Butha-Buthe district, 12 cases of glycosuria were discovered. Seven of these were found to have blood-glucose levels above 140 mg. per 100 ml., i.e. 0·23%. Survey findings in the United States of America indicate that the incidence of diabetes mellitus is 1·7% of the population, i.e. 51 diabetics would be expected in a population of 3,000.

STUDIES IN RED-CELL METABOLISM: I. ALTERATIONS DURING AGING

DR. RALPH E. BERNSTEIN, *South African Institute for Medical Research, Johannesburg*

Disintegration of circulating erythrocytes after a normal life span of approximately 120 days has been attributed to extra- and intracorpuscular factors. Until recently, impairment of the intrinsic metabolism of the red cell had received little attention as a possible initiating cause of disintegration. The metabolic energy of the erythrocyte is derived from glycolysis and is used to maintain membrane function (permeability and osmotic properties) and to prevent the auto-oxidation of haemoglobin to a non-

oxygen carrier. It was thus of interest to investigate whether alterations in the whole process of glycolysis and the activity of critical enzymes and co-factors concerned in glycolysis occurred with *in vivo* aging of erythrocytes.

Red cells from cases with reticulocytosis were separated by centrifugation into younger and older age fractions. The old cells showed decreased glycolysis and a loss of high-energy phosphates and certain glycolytic enzymes. The diminished hexokinase and aldolase enzyme activities were the cause of the rate-limitation in glycolysis.

The decrement in metabolic energy of old red cells was associated with lessened active transport of electrolytes and a diminished ability of the red cell to stabilize its volume against variations in membrane permeability. Decreased glycolysis in the aged cell was postulated to alter membrane function and to lower erythrocytic resistance to mechanical (e.g. buffeting in circulation), chemical (? lytic agent) or other (e.g. phagocytosis) dissolution of the red cell membrane.

MESOTHELIOMAS OF THE PLEURA AND THEIR POSSIBLE ASSOCIATION WITH ASBESTOS EXPOSURE IN THE NORTH WEST CAPE

DR. J. C. WAGNER, *Pneumoconiosis Research Unit, Johannesburg*

Mesotheliomas of the pleura are regarded as uncommon tumours.

During the last four years 33 cases of diffuse mesothelioma have been diagnosed on histological examination of biopsy or autopsy material. Of these patients 28 have had some association with the Cape asbestos fields. Twenty-five were born or spent part of their early years in the vicinity of the asbestos mines. Three others either worked in the mines or transported asbestos as did 7 who were born in the district. In addition 4 further cases, who had no association with the district, had been subject to an industrial exposure to asbestos dust. Two of these were employed in insulating locomotive boilers; one man made asbestos clothing for fire fighting and another was employed in maintaining and insulating steam pipes in a factory in the South West Cape. No definite history of exposure to asbestos has been established in the remaining case. This investigation has been carried out in collaboration with Dr. C. A. Sleggs and Mr. P. Marchand and with the assistance of the staff of the Pathology Division of the Pneumoconiosis Research Unit.

THE LATE SUCROSE FERMENTING PROPERTY OF *PROTEUS MIRABILIS*

DR. T. G. SACKS, *Department of Microbiology, University of Pretoria*

The late sucrose fermenting property of *Proteus mirabilis* is due to the selective overgrowth of the original population by sucrose-fermenting mutants capable of exploiting the extra source of energy. Mutation rates for 3 different strains of mirabilis range from 1×10^{-9} to $1{\cdot}4 \times 10^{-8}$/bacterium/generation.

LYSOGENY IN *PROTEUS HAUSERI*

PROF. J. N. COETZEE, *Department of Microbiology, University of Pretoria*

The incidence of lysogenicity amongst 23 different strains of *Proteus hauseri* was investigated. Three methods of induction were used and a total of 49 indicator strains were employed. Approximately 50% of strains proved lysogenic.

OFFICIAL ANNOUNCEMENTS : AMPTELIKE AANKONDIGINGS

THE MEDICAL ASSOCIATION OF SOUTH AFRICA

(Incorporated in the Union of South Africa)

CIRCULAR TO MEMBERS

and

NOTICE OF AN EXTRAORDINARY GENERAL MEETING

WHEREAS it has been felt for some time that Article 23 (*b*) lacks clarity in that it could confuse the holding of the Association's Annual General Meeting with the holding of a South African Medical Congress,

AND WHEREAS Article 25 makes it obligatory to hold a South African Medical Congress annually while in fact this is no longer the case,

NOW THEREFORE notice is hereby given that an Extraordinary General Meeting of members of the Medical Association of South Africa will be held in the Board Room, Medical House, 35 Wale Street, Cape Town, on the nineteenth day of February 1960, at 3 o'clock in the afternoon, for the purpose of proposing for consideration and considering, and, if deemed fit, of passing with or without modification or addition in the manner required for the passing of a special resolution in terms of the Companies Act 1926, as amended, of the Union of South Africa, the following resolutions as special resolutions, namely:

That the Articles of Association of the Association be and are hereby amended as follows:

I. That *Article* 23 (*b*) be and is hereby cancelled and deleted and that the following new Article 23 (*b*) be and is hereby substituted therefor:

'23 (*b*). The reception of such addresses, and especially the address to the Association of the President, as the Council shall have arranged to be received at such meeting.'

II. That *Article* 25 be and is hereby cancelled and deleted and that the following new Article 25 be and is hereby substituted therefor:

'25. The Council shall from time to time arrange meetings or conferences, alone or in conjunction with other bodies, which it shall be open to every member of the Association to attend, for the purpose of receiving addresses or other communications

DIE MEDIESE VERENIGING VAN SUID-AFRIKA

(Ingelyf in die Unie van Suid-Afrika)

OMSENDBRIEF AAN LEDE

en

KENNISGEWING VAN 'N BUITENGEWONE ALGEMENE VERGADERING

NADEMAAL daar al vir 'n tydlank gemeen is dat Artikel 23 (*b*) onduidelik is deurdat dit verwarring kan veroorsaak ten opsigte van die hou van die Vereniging se Jaarlikse Algemene Vergadering met die hou van die Suid-Afrikaanse Mediese Kongres,

EN NADEMAAL Artikel 25 dit verpligtend maak om die Suid-Afrikaanse Mediese Kongres jaarliks te hou terwyl dit in werklikheid nie meer die geval is nie,

SO IS DIT dat hiermee kennis gegee word dat 'n Buitengewone Algemene Vergadering van lede van die Mediese Vereniging van Suid-Afrika in die Raadsaal, Mediese Huis, Waalstraat 35, Kaapstad, op die negentiende dag van Februarie 1960, om 3-uur in die namiddag, gehou sal word, met die doel om die volgende besluite vir oorweging voor te stel en te oorweeg en, indien dit raadsaam geag word, met of sonder verandering of toevoeging aan te neem, op die wyse wat vir die aanname van 'n spesiale besluit kragtens die Maatskappywet 1926, soos gewysig, van die Unie van Suid-Afrika vereis word, naamlik:

Dat die Statute van Oprigting van die Vereniging hierby verander, gewysig en uitgebrei word soos volg:

I. Dat *Artikel* 23 (*b*) hierby gekanselleer en geskrap word en dat dit deur die volgende nuwe Artikel 23 (*b*) vervang word:

‚23 (*b*). Die ontvangs van sodanige toesprake, en veral die toespraak voor die Vereniging van die President, as waarvoor die Raad die ontvangs of bespreking op so 'n vergadering gereël het.'

II. Dat *Artikel* 25 hierby gekanselleer en geskrap word en dat dit deur die volgende nuwe Artikel 25 vervang word:

‚25. Van tyd tot tyd moet die Raad, alleen of in samewerking met ander liggame, vergaderings of konferensies reël wat elke lid van die Vereniging sal mag bywoon en waarop referate gelewer en ander mededelinge rakende die geneeskunde en ander wetenskappe gedoen kan word, en vir die bespreking van sodanige onderwerpe in verband daarmee as wat die Raad mag goedvind;

relating to the medical or allied sciences, and discussing such subjects pertaining thereto as the Council may think proper, and the Council may arrange to hold such meetings or conferences within or without the Union of South Africa. When held in the Union of South Africa, one such meeting shall be convened at the same place as the Annual General Meeting of the Association and in connection but so as not to conflict therewith.'

Notice is hereby further given in terms of Section 64 of the Companies Act 1926—

(*a*) That if less than one-fourth of the total votes be present at the meeting, it shall stand adjourned to the same day in the following week at the same time and place.

(*b*) That at the adjourned meeting the members present in person or by proxy may deal with the business for which the original meeting was convened, and a resolution passed by not less than three-fourths of such members shall be deemed to be a special resolution notwithstanding that less than one-fourth of the total votes of the Association are represented at such adjourned meeting.

By Order of the Council,

A. H. Tonkin
Secretary

Medical House
35 Wale Street
Cape Town
19 January 1960

die Raad kan dit reël dat sodanige vergaderings of konferensies binne of buitekant die Unie van Suid-Afrika gehou word. Wanneer dit in die Unie van Suid-Afrika gehou word, moet een sodanige vergadering op dieselfde plek as die algemene jaarvergadering belê word en in verband daarmee maar sonder om daarmee te bots.'

Kennis word hierby verder gegee kragtens Seksie 64 van die Maatskappywet 1926—

(*a*) Dat as op die vergadering minder as een-vierde van die totale stemme aanwesig is, sal die vergadering verdaag word tot dieselfde dag in die volgende week op dieselfde tyd en plek.

(*b*) Dat op die verdaagde vergadering die lede wat persoonlik of by volmag aanwesig is die sake waarvoor die oorspronklike vergadering belê was, mag afhandel en 'n besluit wat deur nie minder nie as drie-vierdes van sodanige lede aangeneem is as 'n spesiale besluit beskou sal word, nieteenstaande dat minder as een-vierde van die totale stemme van die Vereniging op sodanige verdaagde vergadering verteenwoordig word nie.

Op Las van die Raad,

A. H. Tonkin
Sekretaris

Mediese Huis
Waalstraat 35
Kaapstad
19 Januarie 1960

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held at the Pretoria Municipal Recreation Club, Louis Botha Avenue, Riviera, Pretoria, on 3, 4 and 5 March 1960, commencing at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
15 January 1960

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word in die Ontspanningsklub van die Pretoriase Munisipaliteit, Louis Botha-laan, Rivieria, Pretoria, op 3, 4 en 5 Maart 1960, begin 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-Penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee was.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
15 Januarie 1960

THE BRITISH COUNCIL

CARDIAC SURGERY : COURSE NO. 032 : LONDON, 20 MARCH–2 APRIL 1960

This, the first course in cardiac surgery to be organized by the British Council, is designed to demonstrate recent advances as well as standard practices in British cardiac surgery to specialists in this field from overseas. Because of the close relationship between medicine and surgery in the treatment of cardiac disease, applications to attend the course are invited from both cardiac surgeons and cardiologists.

The course will be directed by Mr. W. P. Cleland, M.R.C.P., F.R.C.S., Surgical Sub-Dean, Brompton Hospital, and will be based on the Institute of Diseases of the Chest, Brompton Hospital, London, where lectures, demonstrations and panel discussions will be held. In addition, visits to other London hospitals to see pecific operations will be arranged.

Members will assemble on the evening of Sunday 20 March for an opening dinner at the hotel in London at which they will be staying.

Towards the end of the first week the course will move on to Oxford where the work of the Surgical Unit at the Radcliffe Infirmary will be observed. The week-end will be spent in Oxford and will give members an admirable chance to see this interesting university city and the surrounding countryside. From Oxford the course will move to Birmingham and will spend 1½ days with the Department of Surgery at the Queen Elizabeth Hospital before returning to London for the final days. The course will end on the afternoon of Friday 1 April but hotel accommodation will be booked for that night.

Qualification of members. This course, which is arranged for overseas visitors, is open to surgeons specializing in cardiac surgery and to cardiologists especially interested in problems of cardiac surgery.

Numbers. Not more than 20 members can be admitted to this course.

Accommodation. Members will be accommodated in a central London hotel and in hotels at Oxford and Birmingham.

Fee: £47 0s. 0d.

General Information

Duration of course. Members will assemble at the course centre on the afternoon of the first date shown and will disperse before lunch on the last date, unless anything to the contrary appears in the joining instructions.

Proficiency in English. Members of the courses must be proficient in English. They should be able to follow and take part in English conversation conducted at the normal rate. This is essential if they are to derive the maximum benefit from attendance.

Applications. Applicants should apply to the Director, Courses Department, The British Council, 65 Davies Street, London, W. 1.

Registration fee. A Registration Fee of £2 for each course is payable by every candidate on acceptance. The fee should reach Courses Department, London, not later than 1 month from the date of notification of acceptance. The Registration Fee will be considered as part of the Course Fee, the balance of which will be payable on the candidate's arrival at the course centre. No place can be held for more than 1 month unless the Registration Fee is received. A candidate who withdraws from a course for any reason whatsoever forfeits his or her Registration Fee.

Travel to and from the United Kingdom. Members must make their own travel arrangements to and from the course centre. Return reservations should be made, if possible, before members leave their own country as it may be difficult to secure them while in Britain.

Accommodation. No provision is made for accommodation before or after the course. Those requiring such accommodation should book through a travel agent. In the event of real difficulty, The British Council may be able to assist if application is made in writing at least 3 weeks beforehand. Any such application must be accompanied by a deposit of £1 for each night's accommodation required. In any event no guarantee can be given that accommodation will be available.

Course fee. The advertised fee includes the cost of board and lodging, lectures and excursions during the course.

Joining instructions. These will be issued by the Courses Department, through Representatives, giving directions for reaching the course centre, including postal address and telephone number of the centre and latest time of arrival.

Cancellation of courses. The British Council reserves the right to cancel any course, without notice and without indemnity, subject to the return of any Registration or Enrolment Fee already paid.

MINUTES OF THE ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA

HELD IN ST. SAVIOUR'S HALL, ST. PETER'S ROAD, EAST LONDON, ON THURSDAY 24 SEPTEMBER 1959 AT 12.30 P.M.

The President (Dr. R. Schaffer) was in the Chair, and 52 other members were present. Also present were certain members of the South African Medical and Dental Council.

On behalf of the Association, Dr. Schaffer welcomed the President and members of the South African Medical and Dental Council to the Meeting, saying that their attendance was greatly appreciated.

1. *Notice Convening the Meeting*, which had been published in the *Journal* of 15 August 1959, was taken as read.

2. *Minutes of the Annual General Meeting and Adjourned Annual General Meeting held in Pretoria on* 1 *October* 1958 were taken as read. It was proposed by Mr. Armitage, seconded by Mr. Sweetapple and agreed that they be confirmed. The Minutes were signed by the President.

3. *Annual Report of Chairman of Council*, published in the *Journal* of 25 July 1959: The Chairman of Council, Dr. J. H. Struthers, proposed that his Report be taken as read and adopted. This was seconded by Mr. J. D. Joubert and carried.

4. *Financial Statement and Balance Sheet for the Year Ended* 31 *December* 1958, published in the *Journal* of 9 May 1959: It was proposed by Dr. Struthers, seconded by Mr. Joubert and resolved that this be adopted.

5. *Election of Auditors:* It was proposed by Mr. Joubert, seconded by Dr. Lewis S. Robertson and resolved that Messrs. Gurney, Notcutt & Fisher be reappointed auditors for the year 1960, at the remuneration of £250 per annum.

6. *Induction of President:* The retiring President, Dr. Schaffer, stated that he wished to thank the members of Federal Council for the honour which they had bestowed on him by having elected him as President, an honour which he had greatly appreciated.

Dr. Schaffer then proceeded to the induction of the incoming President, Dr. P. F. H. Wagner. He referred to his long and happy personal relationship with Dr. Wagner and spoke appreciatively of his devoted service to the Association and the profession over many years. Amid acclamation he then installed Dr. Wagner as President of the Association and wished him all success during his year of office.

Dr. Wagner took the Chair and thanked the members for the high honour bestowed on him in his election to the Presidency, stating that he would do all that was in his power to foster the interests of the Association.

He extended a welcome to those who would be attending the 42nd South African Medical Congress in East London during the following week.

On behalf of the Association, Dr. Wagner then expressed the members' sincere thanks and appreciation to Dr. Schaffer for the able and dignified manner in which he had carried out his duties as President during the past year. Acclamation.

After Dr. Schaffer's reply, the President thanked members for their attendance and declared the meeting to be adjourned until 8 p.m. on Monday, 28 September 1959.

ADJOURNED ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA HELD IN THE CITY HALL, EAST LONDON, ON MONDAY 28 SEPTEMBER 1959 AT 8 P.M.

A large audience of members and their wives, together with overseas guests who were attending the 42nd South African Medical Congress, was present.

Opening of Congress: After the entry of the platform party, the Rev. Mr. J. Wesley Allen, the President's Chaplain, led the Meeting in prayer.

The President then introduced the Administrator of the Cape, Dr. the Hon. J. H. O. du Plessis, who addressed the Meeting and formally opened the Congress.

The Mayor of East London, Councillor W. P. Osmond, welcomed members of the Association and their families, as well as the distinguished overseas guests, who were attending the Congress.

A short musical interlude followed.

Presentations and Awards

Dr. Wagner presented the insignia of Immediate Past President to Dr. R. Schaffer, who in turn presented the badge of office of President's Lady to Mrs. Wagner.

Association's Medals: Citations were read regarding the recipients, and the President presented the following awards:

Gold Medal to Dr. T. Shadick Higgins, of Cape Town, for distinguished services to the profession.

Silver Medal to Dr. James Gear, of Johannesburg, for distinguished services to medical science and humanity.

Bronze Medals to Dr. Seymour Heymann and Dr. Lewis S. Robertson, both of Johannesburg, for meritorious service to the Association. The Bronze Medal awarded to Dr. P. F. H. Wagner was presented to him by Dr. R. Schaffer, the Immediate Past President.

The Bronze Medal was also awarded posthumously to the late Dr. J. N. W. Loubser, of Bethlehem.

The Hamilton-Maynard Memorial Medal for the year 1958 was awarded to Dr. David Ordman, of Johannesburg, for his article entitled 'Allergy in childhood: Its pattern, control and significance in adult prophylaxis' which had been published in the *Journal* of 5 April 1958.

The Leipoldt Memorial Medal for the year 1958 was awarded to Dr. G. F. C. Troskie for his article entitled 'Kisteuse pneumatose van die peritoneum' which had been published in the *Journal* of 15 February 1958.

Presidential Address: Dr. Wagner then delivered his Presidential Address entitled 'The training of the general practitioner'.*

At the conclusion of his Address, the President introduced the distinguished visitors from overseas to the audience.

The Meeting ended at 9.30 p.m. and was followed by a reception at which the guests were received by the President and Mrs. Wagner.

* Published in the issue of the *Journal* for 3 October 1959 (33, 825).

UNIVERSITY NEWS : UNIVERSITEITSNUUS

UNIVERSITY OF CAPE TOWN : DEPARTMENT OF EXTRA-MURAL STUDIES

PROPOSED PUBLIC LECTURE COURSES IN MEDICINE FOR 1960

The Department of Extra-mural Studies of the University of Cape Town has organized a series of public lectures in medicine to take place during 1960. The lectures will be held in the Physiology Lecture Theatre, Medical School, Observatory, Cape, on Thursday evenings at 8.15 p.m., and are open to all interested members of the public. Admission is 2s.

PART I

Physiology: How the Human Body Works—Dr. A. G. Ramsay

3 March	What its all about
10 March	Muscles and nerves: what they are and what they do
17 March	The heart and the circulation of the blood
24 March	The lungs, breathing, and gas exchange
31 March	What to eat and why
7 April	The transport and digestion of food
14 April	The elimination of wastes from the body and control of temperature
21 April	The controlling systems of the body: (i) The brain and nervous system
28 April	The controlling systems of the body: (ii) Glands and hormones
5 May	Reproduction of the species

PART II

Isotopes—Prof. A. W. Sloan

12 May	Biological uses of isotopes
19 May	Medical uses of isotopes

Biochemistry—Prof. J. E. Kench

26 May	Continuous chemical change in the body
2 June	Genes and proteins
9 June	The biochemical effects of heavy metals
16 June	Pigments in Man
23 June	Biochemical evolution

Diet

28 July	Diet in relation to health and disease—Prof. J. F. Brock
4 August	Dietary fat and coronary heart disease—Dr. B. Bronte-Stewart.
11 August	Diet in obesity ('Why be fat?')—Dr. R. Hoffenberg *or* Dr. W. P. U. Jackson
18 August	Some important conditions in which diet plays some part: (i) Diabetes, and (ii) rheumatism and gout—Dr. R. Sougin-Mibashan
25 August	Diseases of the kidneys—Prof. L. Eales.

Pregnancy and Childbirth

8 September	Pregnancy—Dr. F. Benjamin
15 September	Childbirth—Dr. H. Mukheibir

Anaesthesia

22 September	'Will it hurt, Doctor?'—Dr. A. B. Bull
29 September	'I want to be asleep, Doctor'—Dr. G. G. Harrison
6 October	'You and your anaesthetist'—Dr. C. S. Jones

Immunization

13 October	Immunization against poliomyelitis—Prof. A. Kipps

Alcoholism—Prof. R. Turner

20 October	Forensic considerations
27 October	Psychiatric considerations.
3 November	Medical considerations.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Seymour Heymann, of Johannesburg, has recently been elected to the Fellowship of the American Academy of Pediatrics in recognition of his many contributions to the health and welfare of children.

* * *

South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-Group will be held on Tuesday 2 February in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Mr. A. Gonski will speak on 'Neurosurgery in childhood'. Visitors will be welcome.

* * *

Gedissemineerde Sklerose. 'n Suid-Afrikaanse navorser wil graag al die bekende gevalle van gedissemineerde sklerose in hierdie land naspoor. Om die ondersoek te vergemaklik word alle dokters, wat van pasiënte met die siekte weet, versoek om die informasie aan die Redakteur, *Suid-Afrikaanse Tydskrif vir Geneeskunde*, Posbus 643, Kaapstad, te stuur. Hierdie informasie sal dan aan die navorser wat die ondersoek onderneem, gestuur word, en hy sal dan self nadere inligting oor die pasiënte inwin by die persone wat die informasie verstrek het.

* * *

Benevolent Fund of the Medical Association of South Africa. The Management Committee of this Fund has received a contribution of £36 from the Ladies' Committee of the Medical Benevolent Fund of the Southern Transvaal Branch of the Medical Association, which is under the convenership of Mrs. W. Girdwood. The total amount contributed by this Committee to the Benevolent Fund for the year 1959 was £2,608 3s. 1d.

PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

PETERSEN HOLDINGS LIMITED

Mr. F. J. H. le Riche has been appointed Managing Director of Petersen Holdings Ltd. which controls the pharmaceutical interests of the Federale Volksbeleggings group, i.e. Sana Ltd. and Petersen Ltd.

The controlling interest in Petersen Holdings was recently acquired by Federale Volksbeleggings Beperk from Merchants Industrial Corporation.

Mr. le Riche, M.Sc. *cum laude*. (Stellenbosch), A.R.I.C., was formerly with Klipfontein Organic Products Corporation as Sales Manager.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

PROPCO

British Drug Houses announce the introduction of Propco tablets, a new analgesic preparation, and supply the following information:

Composition. Each tablet contains: 200 mg. of paracetamol, and 200 mg. of salicylamide.

Indications. For the relief of pain and reduction of fever in influenza, arthritis, fibrositis, lumbago, earache, toothache and menstrual pains.

Advantages. Paracetamol is the first major metabolic product of phenacetin, but its administration does not give rise to the formation of methaemoglobulinaemia as sometimes occurs with phenacetin. Paracetamol appears, therefore, to be preferable to phenacetin in clinical use. Salicylamide is less toxic than aspirin; it is not hydrolysed to free salicylic acid and thus causes less gastric irritation.

Dosage. Adults 2 tablets every 4 hours. Children under 12—half the adult dose. No special precautions are necessary.

Packing. Cartons of 12, bottles of 25 and 100 tablets.

SICCOLAM B

British Drug Houses announce the introduction of Siccolam B, a preparation similar to their existing product Siccolam, but with milder dehydrating properties, and supply the following information:

Composition. Siccolam-B contains titanium dioxide, zinc oxide, chlorphenesin and kaolin in a fat-free base. The percentage

of the active ingredients is however smaller for Siccolam-B than for Siccolam.

Action. Siccolam-B rapidly absorbs serum exudates and acts also as a mechanical protective and a barrier to light.

Indications. Exudative skin lesions, e.g. exudative dermatitis and contact dermatitis; varicose eczema; seborrhoeic and infantile eczema; intertrigo, photosensitivity dermatitis, and bed sores.

Generally, Siccolam should be employed in the acute stages of these conditions but Siccolam-B will be found most useful after the severe exudatory phase and it may be preferred from the onset in those conditions in which the inflammatory exudate is not too pronounced. Siccolam-B spreads easily over the affected area and is suitable, when necessary, for prolonged use.

Packing. Siccolam-B is available in tubes of 40 g.

DISTAQUAINE V-K SUSPENSION

British Drug Houses announce the introduction of Distaquaine V-K Suspension, the latest advance in oral penicillin therapy, presenting for the first time potassium penicillin V as a *ready-prepared* suspension, and supply the following information:

Potassium penicillin V is already widely prescribed in the form of Distaquaine V-K tablets. Now the convenient ready-prepared suspension provides an alternative which will be appreciated by young children and those who find difficulty in swallowing tablets.

Indications. All infections due to penicillin-sensitive organisms, except when oral therapy is unacceptable (e.g. because of intractable vomiting). Distaquaine V-K Suspension may be successfully used even in many conditions formerly reserved for parenteral therapy.

Dosage. Adults 125 - 250 mg. 4 hourly depending on the severity of the condition and the response obtained. Children (12 years and under) 60 - 125 mg. 4 hourly. For optimum response the preparation should be taken half an hour before meals (or at least 3 hours after meals).

Presentation. Distaquaine V-K Suspension is smooth in consistency, pleasantly flavoured and available in bottles of 60 ml. containing 125 mg. of penicillin V (as potassium salt) in each 5 ml. dose.

Distaquaine V-K Suspension is manufactured by The Distillers Company (Biochemicals) Ltd. London, and further information on the product may be obtained from the sole importers in the Union and South West Africa: British Drug Houses (South Africa) (Pty.) Ltd., P.O. Box 372, Johannesburg.

Distaquaine V-K Tablets

Distaquaine V-K Tablets are now issued in 3 strengths: 60 mg., 125 mg. and 250 mg.

Packings. The 60 mg. strength is available in bottles of 30 and 200 and the 125 and 250 mg. strengths in cartons of 12, 100 and 500.

Prices are the same as for Distaquaine V.

CORRESPONDENCE : BRIEWERUBRIEK

SEMINAR FOR SURGEONS TO BE HELD IN ISRAEL

The following is a copy of a letter from the Consulate General of Israel addressed to Mr. M. Arnold, Hon. Secretary, Southern Transvaal Branch of the Medical Association of South Africa, dated 31 December 1959:

'We have been advised that a Seminar for surgeons will be held in Israel from 21 to 31 May 1960 under the auspices of the Ministry of Health.

'We understand that the 12th Congress of the International College of Surgeons will be held in Rome from 15 to 18 May of the same year.

'We would be most grateful if you could advise us how best to publicize this function which will take place in Israel among the surgeons of Southern Africa and the Rhodesias.

'Organized tours for 3 days will be arranged for the participants at the close of the Seminar.

Atara Resnekov
Administrative Officer'

STERILIZATION FOR SEXUAL OFFENCES

To the Editor: Dr. Steenkamp[1] posed a difficult problem, but the matter can be disposed of rapidly since other writers[2] have discussed the matter from a variety of angles. It remains, however, to be said that if a State has the right to take life where life has been taken, it certainly has the right to mutilate where sexual crimes are concerned.

What appears not to have been appreciated sufficiently by the previous writers[3,4] is the fact that orchidectomy will certainly not cure the sexual offender, because the so-called 'master gland' is the hypothalamus. Perhaps not in the near future, but some time, one may be able to effect selective destruction of this centre stereotactically.

If the authorities are determined to do something about sexual criminals, it will be necessary for the medical fraternity to supply the answer to how it can be done with safety and with the least amount of mutilation and, what is most important, effectively.

By diverting the attention from the testicles, one can accomplish all that is required by simple ligation of the pudendal arteries[5] or sympathectomy, which will abolish erection permanently.

How can a man commit murder if he cannot lift his arm?

M. J. Joubert

'Bon Repos'
2511 Marine Drive
Brighton Beach, Durban
16 January 1960

1. Correspondence (1959): S. Afr. Med. J., **33**, 572.
2. *Idem* (1959): *Ibid.*, **33**, 655 and 656.
3. *Idem* (1959): *Ibid.*, **33**, 675, 848 and 1025.
4. *Idem* (1960): *Ibid.*, **34**, 80.
5. Leriche, R. (1940): Presse méd., **48**, 1.

PAIN IN STERNUM AND THORACIC VERTEBRAE

Aan die Redakteur: Die siektebeeld beskryf deur *Puzzled*[1] stem grootliks ooreen met wat in hierdie praktyk (ook in die Oostelike Vrystaat) bekend staan as die ‚ka mona'-sindroom.

Ons het die verskynsel tot dusver egter uitsluitlik by Naturelle aangetref en die rugpyn is nie altyd teenwoordig nie. Die benaming ‚ka mona' is gegee vanweë die optrede van feitlik alle pasiënte wat aan die geheimsinnige ongesteldheid ly: op die vraag ‚ubuhlugu kai?' (waar is die pyn?), word die regter-handpalm horisontaal oor die sternum geplaas en die pasiënt sê ‚ka mona' (net hier). Verdere ondervraging is gewoonlik nutteloos: die pasiënt antwoord óf ‚ja' op alle vrae óf ontken die bestaan van ander kwale ten sterkste. Ondersoek toon óf geen afwykings nie óf siektetoestande wat wissel van salpingitis tot gastritis en anemie.

Behandeling in gevalle waar patologie ontdek word, is gewoonlik maklik en die pyn in die bors verdwyn sonder meer namate die salpingitis of wat ook al opklaar. In gevalle waar geen patologiese toestand gevind word nie, is die behandeling uiters onbevredigend en pogings om simptomatiese verligting te gee deur die toediening van pynverdowende middels, is gewoonlik onsuksesvol. Aan die ander kant lei algemene versterkmiddels in sommige gevalle, alkaliese maagmengsels in ander gevalle, en selfs purgeermiddels en ander onwaarskynlike medikamente in party, tot dramatiese genesing.

Die vraag het dus ontstaan of die pyn waaroor gekla word 'n werklike pyn in die algemeen aanvaarde sin van die woord is. Na my mening is dit nie.

Waar die Blanke ‚hartseer' is as hy treurig voel, is ‚hartseer' vir die Naturel 'n meer praktiese, byna organiese, gewaarwording en, byvoorbeeld by die afsterwe van sy kind, sal hy sê dat dit seer is ‚hier binne' en sy hand op die bors plaas net soos in gevalle van ‚ka mona'-sindroom gedoen word. So ook is die hart seer as die liggaam ongesteld is, d.w.s. die Naturel het pyn agter die sternum afgesien van aan watter siekte hy werklik ly. Soms word die hart ook seer omdat hy bekommerd is en die pasiënt verlang dan net 'n roetine-ondersoek om seker te maak dat alles nog wel is. Die uitspraak dat die hart nog ‚mooi loop' is dan gewoonlik genoegsaam om die pyn te laat verdwyn.

Hoewel ek *Puzzled* se idee van 'n moontlike virussiekte nie summier verwerp nie, is ek tog van mening dat die pyn in die bors waaroor sy pasiënte kla in werklikheid meestal nie bestaan nie en slegs 'n manier is van sê: ‚ek is siek'. Die oorsaak moet elders gesoek word, maar die kanse om dit te vind, is net so gering soos by die Blanke wat ook net sê ‚ek is siek' en verder weier om iets te sê ‚want die dokter is mos slim en moet self uitvind wat verkeerd is'.

Thabo

13 Januarie 1960

1. Briewerubriek (1960): S. Afr. T. Geneesk., **34**, 20.